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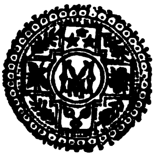

John C. Jay J

1879

A

SYSTEM OF MEDICI

EDITED BY

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PREFACE.

THE fifth and concluding volume of this system of medicine, will, I believe, be as instructive and interesting, as any of those which have preceded it. It contains information upon a very wide range of diseases, and the manner in which the authors of the respective articles which fill its pages have dealt with them throughout, has been such as to justify my belief. To those who have contributed to this volume, and to whom I can now address myself, I offer my heartiest thanks, both for the readiness with which they have done their work, and for the industry, learning, and critical faculty which they have displayed.

One of the contributors, Dr. Basham, was removed by death before he had completed his work, but I was most kindly and ably assisted by Dr. Frederick Roberts, in the anxious work of editing MSS., which in some cases proved to be but half completed. To Dr. Gowers, I am deeply grateful, not only for the very able papers which he has written, but also for his kindness in rendering me very great assistance in the preparation of this volume for the press.

It is not a matter for much surprise that a work, which has taken so long a time to produce, by men who had already become eminent in our profession,—the longevity of which is less than that of most,—

should not be completed until many of its contributors had p beyond the reach of either our praise or our blame, and that should not have seen the work, which they had done so care placed in its proper niche in the edifice that they had helpe raise. To two of these, Drs. Warburton Begbie and Sibson, I made some allusion in my note of introduction to the fourth vol of the others, from whom I had expected further help, I say something here.

Dr. Anstie,—whose loss the profession has never ceased to feel about whom, every one who had the privilege of his frien can scarcely speak in terms that shall do justice to the memory o earnest, loving soul, and of that devotion to duty and science w led to his early death,—was one of my most helpful and consid coadjutors in the production of the second volume, not onl contributing his papers to the *System of Medicine,* but by the assistance which he rendered me in the preparation of the se volume for the press. From him, I had hoped for much more but, for him "there was nobler work to do."

Dr. Basham entered most heartily into the scheme of this book the publication of his papers was long delayed in consequen circumstances which I explained in my note to the fourth vol Those which he has left behind him, nearly completed, and w have been finished by Dr. Frederick Roberts, will shew, as other works have done, the simple and scientific character o writing, and the thorough practicality of all his teaching.

The death of Dr. Warburton Begbie, to whom I have alr alluded, occasioned another of the great losses which this *S* has sustained. There are few, if any, who have surpassed hi the scientific ability, scholarship, and high moral tone of his wor

Dr. Hughes Bennett, whose strong personal views on patholo questions, gave an interest to the subjects with which he dealt, tributed the article on Phthisis, containing much original matter and thought, which will be useful to all interested in the history of tuberculosis.

The death of Dr. Thomas Hillier, the whole of whose published works afforded such rich promise of still more useful labours, occasioned another loss which the profession could ill sustain, a loss also to myself as a friend, colleague, and, as I hoped, future fellow-worker.

To Dr. Parkes, it is absolutely impossible for me to express my obligation. From the first day that I planned the construction of these volumes, until the last day but one before his death, he was my faithful counsellor and friend. His direct contribution to the *System of Medicine* was comparatively small, but the help that he gave me, indirectly, was immeasurably large. He was punctual to an hour, and precise to a three-place decimal. High above all his scientific work, great as it was, rose his grand moral character, for which all who have known him well must be profoundly grateful, in feeling that they are "better men, and are conscious that they may be better still."

Dr. Hyde Salter contributed a valuable paper on Asthma, condensing for this *System* the result of a great many years of good, scientific work. His well-known and recorded personal sufferings from the malady, upon which he wrote so ably, supply an interest and instruction that could perhaps have not been otherwise obtained.

To Dr. Sibson, the profession is much indebted for some most carefully elaborated papers, which appeared in the fourth volume, and to which in its introductory preface I have already alluded, so that I can here do no more than reiterate my regret for the loss, which medical science and literature have sustained in his unexpected death.

Dr. Edward Smith, a highly distinguished member of our profession, who had contributed much to the advance of medical science, and especially in some of its more practical bearings upon daily took a warm interest in the volumes which I have edited, and, his work was prematurely arrested by death, he had hoped to re still further assistance in their production.

Dr. Squarey, a worker of great earnestness and promise, tributed several articles on Diseases of the Mouth and Throat the third volume, but his life was cut short in the midst of devoted labour.

The value of this work is greatly enhanced by the Indice each separate Volume, and also to the five Volumes collectiv For the production of these, I have to thank, and I do so heartily, Mr. Marcus Beck, the late Dr. Loy, and Dr. J. W. Langm the last of whom has furnished the Indices to the fourth and the fifth volume.

To the dead and to the living, I again express my thanks, feel most grateful that I have been able to put into the hands of medical profession a series of admirable original contributions t medical literature of our country, of which I think any co might be proud.

J. RUSSELL REYN

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ON MEDIASTINAL TUMOURS.

BY R. DOUGLAS POWELL, M.D., F.R.C.P.

THE Mediastinum is that central space situated behind the sternum and between the pleuræ which is occupied by the heart and the great vessels connected with it, the trachea and its main divisions, the pneumogastric and phrenic nerves, and the thymus and bronchial glands.

The lungs with their pleural coverings are closely applied on either side of this region, their anterior margins overlapping it in front to an unequal extent on the two sides, so that in health the dulness on percussion that the solid contents of the mediastinum would naturally yield is in a great measure obscured, and permitted to become apparent only at the upper part of the sternum and over the triangular area of the heart's dulness. In emphysema of the lungs the mediastinum may be yet more completely covered up; in other pulmonary affections attended with diminution of bulk, it may become uncovered on one side or on both, giving rise to some singular, and at times perplexing, distortions of the mediastinal dulness without any corresponding disease within the space itself. The division of the interpleural space into anterior and posterior mediastinum is entirely arbitrary, the former term being loosely applied to that portion of the space in front of the trachea and the roots of the lungs, the latter to that portion situated behind this plane. The mediastinum is in health altogether obscured to us from behind by the spine and the thick posterior margins of the lungs closely in contact with it.

Our present concern being only with morbid growths affecting the mediastinum, we shall refer to such other diseases as aneurism, abscess, pericardial effusion, &c.,—only in so far as they affect diagnosis.

VARIETIES, ÆTIOLOGY.—Carcinoma,[1] sarcoma, and lymphoma are the three varieties of morbid growth which may give rise to tumour in the mediastinum. Since the time, only a few years ago, when attention in this country began to be more closely directed to the finer distinctions, based on anatomical structure, which separate true cancers from other tumours, and these from one another—an inquiry first entered upon by Professor Virchow, and largely stimulated moted by the labours of the Morbid Growths Committee Pathological Society of London—the rarity of primary carci the mediastinum has become more evident, and a large pr of the cases that only a short time ago were designated encephaloid, or scirrhous, or soft cancer, would now be classed the sarcomata or the lymphomas. Still, cases of true cancer p affecting the deep-seated parts of the mediastinum do now a occur; *e.g.* one such case is recorded by Dr. C. T. Williams Pathological Transactions, vol. xxiv., its cancerous nature bei firmed, on minute examination, by Mr. Arnott. Even as a se growth, cancer rarely affects the mediastinum, save when, as, h not uncommonly happens, it directly penetrates through th wall from a cancerous breast.

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[1] Throughout this article the terms "carcinoma" and "true cancer" are applied to that form of new growth whose typical structure is met with in the scirrhous breast.

Sarcoma, too, as a primary disease, is most rare in this sit when it occurs it usually arises secondarily to some sim associated growth situated elsewhere. Nor is it always cle a secondary growth arises in the mediastinum; its path of mission is not in all cases evident. We cannot always evidence of a lymphatic connection between the primary and se growths, nor, on the other hand, is it easy to see how a disea could be conveyed from a distant part to the mediastinum t the circulation without first involving the lung; and in view difficulty, it is worthy of remark how frequently the disease, al mainly mediastinal, involves also one lung to a greater or less It is very possible that, in some of these cases, the secondary may have really been conveyed to some portion of the lung or first, and formed a small nodule there; but that, the bronchial becoming early infected, the disease in them proceeds with suc rapidity as soon to outstrip and obscure its pulmonary origin, give the case the clinical and even *post-mortem* features of p mediastinal disease subsequently involving the lung. This expl at least occurred to me as best accounting for a case of osteo-s of the mediastinum and lung, occurring subsequently to the r of a shreddy sarcomatous growth from the knee-joint.[1] The always remain, of course, the possibility of the disease of the m tinum, though secondary in point of time, being due to a recu or a continuance of the same constitutional dyscrasia which the production of the first. Experience has, however, of late gone against the validity of such an hypothesis.

Of morbid growths affecting primarily the mediastinum, *lym* or *lympho-sarcoma*, or, as it is sometimes designated, *lymphade* is by far the most common. It was Dr. Murchison who first nized lymphadenoma as a distinct variety of morbid growth, in of disease affecting the intestines, liver, mesentery and heart which he brought before the Pathological Society of Lond

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[1] Path. Trans. vol. xxiv. p. 28.

November 1868,[1] and of the minute character of which an ample report is appended to his description by the Morbid Growths Committee. In the same volume, p. 102, Dr. Church has also described a case of "Carcinoma of the pericardium, anterior mediastinum, and lymphatic glands, in the thorax and abdomen," which he recognized as different in minute structure from true cancer, and regarded as more correctly "ranked among the mediastinal sarcomatous tumours mentioned by Virchow[2] as approaching so closely to the structure of lymphatic glands as to be with difficulty separable from them." In the next volume, (xxi. of the Transactions, p. 358,) is recorded as such by myself the first case of lympho-sarcoma or lymphadenoma of the mediastinum, although there are many cases in earlier volumes related as instances of cancer which would be undoubtedly more correctly included under the newer term. In subsequent volumes examples of the disease are given by Drs. Murchison, Bennett, Payne, Dickinson, and others.

In most cases, the growth originates in the lymphatic glands, either in the anterior mediastinum or at the root of the lung, the connective tissue surrounding the glands becoming quickly implicated. In a case, however, reported by Dr. Church, and in another by myself, the thymus gland appeared to have been the original seat of the disease. The growth invades other tissues, the neighbouring glands, the lungs, the heart, and even the vessels. It does not, however, incorporate to itself with the same avidity as cancer does all the tissues with which it comes in contact, but prefers to creep along the bronchial or vascular sheaths, and to involve organs more slowly, guided by the lymphatic paths into their interior. The calibre of large bronchi, veins, or even the auricles of the heart, may, however, be invaded by flattened projections of this growth, which by the unaided eye could not be distinguished from cancer. As a local disease, then, lymphoma in this situation is decidedly malignant, but in an intensity rarely as great, and sometimes much less, than that of cancer. It is sometimes, however, a part of a more general disease, affecting more or less the whole glandular system, and in one remarkable case, which was for several months under my observation both as an out-patient and in the wards of the Brompton Hospital, and which subsequently terminated fatally in the Middlesex Hospital under the care of Dr. Murchison,[3] the disease, mainly affecting the glands of the neck, mediastinum, and axilla, and the spleen, was marked by periodical attacks of fever, accompanied by intumescence of all the affected glands and of the spleen, which gave to it an altogether peculiar character. After death, almost every organ was found disseminated with lymphatic growths identical in structure with those already described. In this ca was seen exemplified the highest degree of malignancy conc only comparable to that of disseminated cancer or miliary tuber

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[1] Vide vol. xx. p. 192.

[2] Die Krankhaften Geschwülste, Band ii. p. 376.

[3] A description of the clinical characters presented by this case while in the Middlesex Hospital, with an account of the autopsy and an admirable summary of the literature of the subject, is given by Dr. Murchison in the Path. Trans. vol. xxi. p. 372, and to it is appended an account of the microscopical examination of the diseased structures by Dr. Sanderson.

AGE.—Growths in the mediastinum may be met with at alm period of life, but they are more prevalent before the middle and it is useful to remember this fact, since, if we meet with a mediastinal tumour of doubtful nature in a patient under the 25, it is more likely to be malignant than aneurismal, this prol being increased as the age is earlier. Of six cases which have under my personal observation, all but one were under 30,— at the age of 6, two at 20, one at 27, and one at 29; the six being aged 49. Of seven cases specially referred to by Dr. I as mediastinal, two occurred at the age of 11, one at 17, one one at 23, one at 40, and one at 60. So that if we might fairl an average from such limited numbers we should get 24·8 as th age for the occurrence of this disease.

SEX.—As regards *Sex,* five out of Dr. Bennett's seven cases were females, and five of my six cases were also females. other hand, however, of six cases specially referred to by Dr. Thompson in a pamphlet on Mediastinal Tumours, published i all were of the male sex. We must, then, for the present, s these growths may occur in either sex, with perhaps a sligh ponderance in favour of the female sex. When a mediastinal invades the lung secondarily, it appears to have a special b exclusive preference for the left lung, the other lung as a rule escaping. In four of Dr. Bennett's cases one lung was invade in each instance the left. Of the three of my cases in whi lung was invaded, it was in two instances on the left side.

SYMPTOMS.—The *Symptoms* of mediastinal tumours are lit fluenced by the kind of growth (and the same remark appl physical signs), they are due to compression or obliteration of and nerves, of the air-tubes or œsophagus, or to the invasion heart or the lungs or other structures on the confines of the They therefore closely resemble those presented by aneurismal tu and the diagnosis between the two is often perplexing.

In cases of tumour, deep-seated *pain in the chest* or back so prominent a symptom as in aneurism; there is often no experienced until the growth approaches the surface, when it as the pleuritic character. The characteristic stabbing pain of ca occasionally complained of. If it is borne in mind that mali growths, cancer, lymphoma, sarcoma, differ from aneurismal tu in two important respects,—viz. (*a*) they tend to incorporate to selves the structures they encroach upon, thus invading and rep more, and compressing and displacing less than aneurism; and change in the *direction* of expansion, so common in aneurism, is less so in them, and is not attended with that relief to symptoms dependent on local removal of pressure—it will be more readily understood why the pressure symptoms should *as a rule* be more insidious, yet when present more constant and persistent with them than with aneurism.

The *Dyspnœa* depends upon the size and seat of the tumour, and increases day by day with its growth; but severe paroxysmal attacks of dyspnœa are occasionally witnessed in tumour as in aneurism. These paroxysms are usually due to direct pressure upon the trachea or a main bronchus, and are more frequently observed among the later symptoms of the disease. *Cough*, dry, ineffectual, or attended with only scanty mucous or frothy expectoration, is an early and very constant symptom, and, together with a certain sense of constriction or pain about the sternum, constitutes the complaint for which the patient usually first seeks advice. The cough may have a clanging laryngeal character, and may be attended with *huskiness of voice* or *aphonia*. These symptoms are, however, less frequent than in aneurism. *Sanguineous expectoration* may be present, and is a sign that the tumour has invaded the lung. Profuse *hæmoptysis* is rare, but has been met with as an early symptom. In the later stages of the disease profuse hæmoptysis sometimes occurs, and is followed by marked relief to pressure symptoms; it then becomes an important sign of tumour. In a large proportion of cases, however, no hæmoptysis occurs throughout the disease. *Dysphagia* is in mediastinal growth a far more common and prominent symptom than in aneurism. It is more constant when present, although, as in the case of dyspnœa, it too may be increased by paroxysms, more especially in the earlier stages of the disease.

Physical Signs.—At the time of seeking advice patients with mediastinal tumour are not as a rule emaciated; they often indeed appear to be well nourished, although on inquiry it will be invariably found that they have of late lost flesh; nor do they ever evince as the disease progresses that degree of emaciation so commonly seen in chronic phthisis, save in those cases in which the œsophagus is involved or pressed upon. The face is usually pale, with often some lividity of lips, and in most, perhaps in all instances, there is a certain anxiety of expression, a slight contraction of the brow, giving an aspect of distress which is often sufficient to mark off the case as one not of ordinary chest disease. A slight staring of the eyes, with noticeable puffiness of face, commonly present, may, as the disease advances, be intensified into the aspect of semi-strangulation, characteristic of a tumour pressing upon the great veins. The *temperature*, unless there should be some inflammatory complication, is not raised. In the exceptional case already referred to the periodical attacks of fever were but phases in the progress of a general disease affecting the whole glandular system.

In further considering the physical signs of mediastinal tumour it

must be remembered that, as has already been incidentally ren
these growths very commonly involve sooner or later one
lungs. The lung thus secondarily affected is most frequently,
always, the left.

On inspecting the chest, some alteration in shape is frequentl
observed. The upper sternal region may be unduly prominent,
side of the chest may be both to eye and measurement larger th
other, the enlargement being perhaps more decided above than
the nipple level. The side, however, which yields most evide
disease is not always the larger, it may be smaller, to measure
and this negative sign, taken with other positive ones, *e.g.* di
ment of heart, would be very significant of tumour. Cases
been observed by Dr. Pollock, Mr. Holmes, and others, in whi
tumour has projected through the sternum and cartilages.
enlarged glands at the root of the neck, or in the axilla, mo
adenoma, fixed in cancer, may give us a clue to the nature
disease. The superficial veins of the chest are frequently
distended, more frequently and more decidedly so than in ane
They may be more distended on one side of the median line th
the other, and one upper extremity may show venous obstructi
œdema.

Displacement of heart is one of the most important signs of
thoracic tumour; it is a result of direct pressure, and its dir
is, generally speaking, determined by, and is an important
of, the seat of the tumour. The growth may occupy the uppe
of the anterior mediastinum, and extend downward in front
pericardium, covering it with a thick, solid apron, or, growing
behind, it may push forward the heart against the sternum; or
encroaching forwards from the root of the lung (a common
between it and the pericardium, it may press aside the heart.
should endeavour, therefore, in all cases, by palpation and aus
tion, to ascertain the exact and relative position of both the ape
base of the heart. In certain rare cases—one such came und
notice in Dr. Cotton's wards, at the Brompton Hospital, in 1866
heart is fixed *in situ* by the growth extending on both sides of it
mediastinal growth may extend downwards between the lun
heart to the diaphragm, forming a large mass between it and th
of the lung. Some downward displacement of the *liver* or *stomach*
hardness and bulging of the hypochondrium, are then to be obs

Increased *dulness* on percussion is an essential sign of media
tumour. It may amount to only a patch of lessened resonance a
sterno-clavicular angle or in one interscapular space, or there
be three or four square inches of dulness over the upper sternum
tinuous below with that of the heart area. Again, the toneless
cussion may and often does extend beyond the confines o
mediastinum, so as to include the whole or a greater part of one
in consequence of the growths involving by direct invasion, o
directly, by the destruction of its main bronchus, the lung on one

veral questions in diagnosis arise from this fact, and it is curious w invariably the disease in such cases is mainly one-sided, and how quently the other lung altogether escapes. Supposing the dulness be thus extended, its *quality* and *distribution* become matters of eat importance in diagnosis. Its quality is essentially hard and re- ting—often unequally so at different parts, so as to have a lumpy aracter, being more toneless and resisting over small scattered eas than in the intervening parts. Above the clavicle, in the outer apular region, and at the acromial and axillary regions of the chest, resonant note may still be obtained—in fact, a mediastinal growth, hen it invades a lung, almost invariably does so from its hilus, tending outwards so as to occupy the whole middle part of the ng, coming to the surface in rounded prominences and leaving the moter "corners" of the thorax, so to speak—the summit, humeral, d scapular regions, and the base—to be last involved. It will be membered that in pleural effusion the dulness advances steadily om below upwards, the circumferential parts of the chest first coming toneless, the central—sternal and interscapular regions being last affected. Displacement of heart away from the affected le is common to both conditions. If the base of the lung, however, comes the seat of secondary pneumonia, or is collapsed by some tendant effusion, the validity of the contrast just drawn is somewhat scured.

On auscultation the heart's sounds are found to be unduly con- cted over the dull region in front and too audible in the inter- apular region behind. When the tumour is mainly seated in the terior mediastinum, the conduction of the heart's sounds may be tense in this region, and may be attended with an impulse distinctly preciable to the ear. The *impulse* is, however, in such cases ocking, not expansile; but it sometimes closely resembles that elded by an aneurismal sac thickly lined by coagulum. A systolic *urmur* is sometimes audible over some portion of the dull region; has, however, the simple, short, blowing character, distinct from the sping or expansive bruit which would most likely accompany an eurism of similar superficial dimensions. In cases of tumour owing from the posterior mediastinum, the heart pressed against the ernum may yield *to the ear* a very peculiar sensation, analogous to at experienced by the hand when laid upon a struggling bird. an obscure case which came under my notice three years ago, the esence of this sign, together with displacement of the apex towards e ensiform cartilage, enabled me correctly to surmise the nature of e case before any other positive sign could be detected. The peri- rdium and the heart, more particularly one of the auricles, very ten become involved in the growth, and a *cardiac murmur* or *friction und* may thus be given rise to.

The *respiration* is commonly bronchial over the tumour, or it may ve a stridulous or sibilant character. Stridor is less common than aneurism, and for the reason before named, that growths tend to

occlude rather than compress the tubes with which they c
contact. The observation of very well marked stridor has s
led me carefully to examine for a tumour which has proved
exist, that the sign has, for me at least, lost much of the valu
ascribed to it. Still it would not be safe to disregard it, esp
when localized at one part of the chest. When the growth, h
occupies the anterior mediastinum, and is of considerable thi
no respiratory sound may be audible over it, more or less br
respiration and râles being heard in the outer subclavicular
It is indeed in parts of the chest distant from the tumour
mediastinum that we often get auscultatory signs most sugges
its presence, *e.g.* there may be observed at one base marked
ness of respiration, amounting to a mere muscular struggle w
any accompanying respiratory sound, yet the percussion dul
perhaps little if at all impaired, quite insufficiently so for eff
there is no ægophony, the vocal resonance is diminished, o
gether annulled. On the opposite side the respiratory mur
normal or exaggerated. Here is a grouping of signs very confir
of a tumour compressing or obliterating a main bronchus.

When the dulness extends from the mediastinum over one
the chest, it is accompanied by enfeebled or even annulled
ration, and we may have many of the signs most significant o
effusion into the pleura—displacement of heart, dulness, abse
respiration, and even of vocal fremitus, with enlargement of th
The diagnosis between the two is only made with extreme diff
and, without puncture of the chest, is often indeed impossibl
this dilemma, however, attentive auscultation will often di
here and there over the affected side a slight grating *friction*
a sign which becomes of the greatest importance, showing the p
surfaces still to be in apposition.

Moist rhonchi—mucous or gurgling râles—are never to be he
any abundance over the dull region, as would be almost inevitabl
case in any similar extent of consolidation from scrofulous disea

This remark is not the less true although after death w
occasionally find softened patches in a lung which has be
invaded by a growth, the softening having as a rule been preced
obliteration of the bronchi leading to them, so that they yie
sign during life. Indeed, on making a section of a lung whos
has been invaded by a tumour, we frequently find a striking ap
ance as of multiple abscesses dispersed through the organ, an
condition has been repeatedly so described. In truth, however,
"abscesses" are generally nothing more than bronchial tubes w
have become enormously distended with secretion in conseq
of obstruction at the main bronchus, collapse and slow in
mation of the surrounding pulmonary tissue being also present.
distended tubes, with their opaque contents, shine through the pl
surface of the lung as yellow spots, giving to it a remarkable ap
ance. No doubt some of the alveoli become filled and yield b

the accumulating pressure of the bronchial secretion. Sir George Burrows many years ago related, to the Medico-Chirurgical Society, a case of carcinoma of the lungs in which the right bronchus was obstructed, and some bronchial tubes in different parts of the lower lobe "when cut across were found distended with thick, yellow, tenacious pus, giving the appearance of small abscesses." A typical case of the kind, too, is described by my colleague, Dr. C. T. Williams, in the twenty-fourth volume of the Pathological Transactions, and I have seen other instances, the most remarkable one being from a case of aneurism compressing the left bronchus, of which I made the autopsy in July 1870. An extract from my note-book states as follows:—"Left lung very large, consolidated throughout. Bronchial tubes much congested and dilated, many presenting terminal dilatations, filled with muco-purulent secretion. Lung studded throughout with nodules of yellow pneumonia, having for their centres bronchial tubes which exude their secretion on pressure. Surrounding these nodules the tissue is in a condition of gelatinous pneumonia, so that the total result is a solid lung. Some of the broncho-pneumonic centres have broken down into small cavities filled with muco-purulent fluid. Right lung healthy, but in a state of active congestion."

Dr. Budd[1] regards the secondary inflammatory changes—thickening and adhesion of pleura, and inflammatory destruction of lung—that occur in a lung whose root is invaded by cancer as due, not to irritation of the invading new growth, for cancer *per se* has little tendency directly to cause inflammation of the surrounding parts, nor for the most part to obstruction of veins and arteries (pulmonary and bronchial) which might cause gangrene or atrophy of the lung, but he thinks that these changes result "from the tumour involving and destroying all or great part of the nerves with which the several tissues are furnished." In one of his cases Dr. Budd regards the pericarditis present as due to the same cause. I have seen one case to which this explanation might very well apply. It was one that occurred at the Brompton Hospital under the care of Dr. Pollock in 1868, in which there was found after death a tumour invading the left lung from its root to about one-third of its extent, the rest of the lung being shrunken from inflammatory softening; encroaching upon the summit and also upon the base were found two pleural cavities occupied by purulent fluid: the left main bronchus was almost, but not quite, obliterated by the growth. It is readily conceivable that partial destruction of the pulmonary nerves may, by lowering the vitality of the lung, render it more liable to the occurrence of inflammatory changes, and that complete destruction of these nerves at their origin might directly induce inflammatory destruction of the organ. In the main, however, I should feel disposed to regard the mechanical obstruction at the main bronchus as being, in most cases, sufficient to account for such secondary lesions as we find. In some of the cases which I have quoted the secretion from the br membranes went on with a vigour undiminished by any impa of nervous influence, and the pneumonic changes present apparently, directly due to obstruction to the escape of this sec It is very possible that pressure upon the nerves may give muscular paralysis of the bronchial tubes, as suggested by Dr. B without affecting their secreting power.

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[1] "On some of the effects of Primary Cancerous Tumours within the Chest." Med. Chir. Trans. vol. xlii., 1859.

DIAGNOSIS.—In relating the symptoms and signs of medi tumours in the present chapter and in the succeeding secti aneurism, much has been said incidentally respecting the dia of these tumours from other diseases. We have still, howe summarise and discuss the most important difficulties that ma in the way of diagnosis. These difficulties vary somewhat, acc as the disease is (*a*) purely mediastinal, or (*b*) involves the secondarily.

(*a*.) Growths which are purely or mainly mediastinal may c simulate *aneurism*. The following are the chief considerations would tend to decide the question in favour of the tumour be morbid growth:—

The age of the patient being under 25.
The presence or history of tumours elsewhere.
The absence of marked disease of the vessels.
The absence of characteristic pulsation or bruit, especially combined with
The presence of local venous engorgement, and
Extensive area of superficial dulness.

"Extensive area of dulness must in aneurism mean a large sa with such a large tumour we should almost inevitably get m expansile pulsation. Again, aneurismal sacs, before they produce e sive dulness of any portion of the parietes of the chest, point, were, in some particular direction becoming distinctly prominen producing an eccentric motion around them in consequence o thoracic parietes being absorbed, or yielding at the point of gr pressure."[1]

In the early stages, however, of the disease, it may be extre difficult, nay, impossible, to make the diagnosis with certainty. must duly consider and weigh the probabilities in each case, being no further rules of sufficient general value to be wort mention here.

Mediastinal abscess is very rare, at least of such dimensions simulate tumour. Such an affection would usually be accompa with scrofulous abscesses elsewhere, and probably with hectic s toms.

Sub-sternal thickening may cause many of the signs of tu particularly when associated, as in one case which has been for time under my notice, with œsophagial spasm. The effect of treatment (antisyphilitic) upon such cases soon discovers their real nature.

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[1] Graves' Clinical Lectures, 1848.

A variety of chronic pericarditis has been described by Prof. Kussmaul[1] under the name of *callous mediastino-pericarditis,* in which the pericardium becomes greatly thickened and its cavity completely obliterated by the tough products of a chronic inflammation, which, moreover, extends to the cellular tissue of the mediastinum, indurating it and surrounding the great vessels with a contractile tissue which constricts and distorts them. The increased mediastinal dulness and other signs attendant upon this condition closely simulate those of tumour, but Kussmaul states that there are two signs which are characteristic of this lesion,—viz., a complete, or almost complete, failure of the radial pulse during inspiration, and, simultaneously, visible swelling of the great veins of the neck instead of the collapse that usually takes place during this portion of the respiratory act. Adhesion of the great vessels to the sternum, either directly or through the medium of the pericardium, is supposed to account for these phenomena.

Certain cases of *phthisis,* in which chronic disease at one apex has led to exposure of the mediastinum from retraction of the margin of the lung on one side, may be mistaken for mediastinal disease. Such cases, however, especially some cases of so-called senile phthisis, are more apt to be confounded with cancer of the lung or with aneurism. They have already been referred to in the previous chapter.

(*b.*) A mediastinal growth secondarily invading the lung on one side may present more difficulties in the way of diagnosis than one more strictly confined to its original site. Deeply seated, invading the lung at its hilus, and, as it were, functionally choking it, such a tumour may simulate *chronic pneumonia,* local or general *empyema,* or *aneurism* of the descending aorta.

The close resemblance between certain cases of mediastinal growth spreading through a lung and effusion into the pleura has already been referred to. The signs in favour of tumour may be thus summarised:—

1. An uncomformity of increased measurements to those which would be occasioned by fluid accumulation.
2. The presence of large tortuous veins and œdema of upper extremities or head.
3. Dulness marked at the mediastinal region becoming less uniform in tone and firmness at the circumferential parts of the chest, where patches of resonance may be found which could hardly co-exist with fluid.
4. The loud transmission of the heart's sounds (Walshe).
5. The detection of pleuritic friction-sound over parts dull on percussion. (It must be remembered that there may be some effusion

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[1] Berliner klinische Wochenschrift, 1873, Nos. 37, 38, and 39. An abstract of these papers is given by Dr. M. Bruce in the Medical Record for December 17th, 1873.

which has supervened upon the mediastinal disease, and tl effusion may be general or limited.)

6. Hæmoptysis or "currant-jelly" expectoration would nega disease, being one simply of effusion.

7. The presence of the signs of pressure upon central parts (V

This last-named consideration is certainly of value, but it m lead; *e.g.* I have myself seen two instances in which consi effusion into the pleura has been attended with that peculiar la cough and husky voice which I regarded as significant of tuı addition to the effusion, and which in one case led others o experience also into the same error. These signs both, howev appeared after the removal of a large quantity of purulent Another pressure sign I have seen exemplified in a case of effusion, which might suggest some associated mediastinal t though more probably of aneurismal than malignant kin increased size, tortuosity, and throbbing of the radial and b arteries on the *affected* side. The case was under the care colleague, Dr. Tatham, and the best conclusion we could come that the phenomenon was due to hardening and hypertrophy vessels from increased resistance to circulation through them, sequence of impediment to venous return from the limb. The no œdema of the limb, however, and after the removal of th the thickening remained; no sign or symptom of tumour ha occurred.

8. In all cases of doubt, and when the dyspnœa is at all urg exploratory trocar should be inserted.

Cases of mediastinal growth invading the lung from its roc often been mistaken for *chronic pneumonia.*

Dr. Walshe lays stress upon the following signs as distingı tumours from chronic exudative pneumonia:—[1]

1. A tendency to increase instead of diminution of bulk affected side.

2. Implication of the mediastinum.

3. The more serious change in the results of percussion.

4. Emaciation is of earlier appearance and more marked in c pneumonia than in tumour.

5. Dyspnœa out of proportion to extent of consolidation f the diagnosis of tumour.

These five signs would be of equal value in distinguishing be chronic pneumonia in its less restricted sense of chronic inflamı consolidation of the lung affecting the lower lobe, and medi growths secondarily affecting the lung. Dr. Walshe gives three f distinctions, viz.:—

6. The failure or disappearance of vocal fremitus, which rema chronic pneumonia;

7. The different characters of respiration in the two diseases

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[1] Diseases of the Lungs, 4th Edition.

8. The presence of hæmoptysis and red jelly-like expectoration which never occur in chronic pneumonia. These do not, however, help us in eliminating those chronic basic consolidations which do not clear up, and which are therefore most apt to come before us for diagnosis from tumour. I have for instance so repeatedly seen cases of chronic pneumonia in which from great thickening of the adherent pleura the vocal fremitus has been much deadened or almost annulled, that I cannot but regard the distinction No. 6 as apt to mislead. The respiration, too, in such cases is remarkably feeble. The diagnosis would, under such circumstances, be cleared up in favour of tumour by finding with these signs tolerably limited to the lower lobe the *heart displaced towards the opposite side.*

I have had also under my observation for some years a patient with consolidation of the base of the right lung who has had decided hæmoptysis on several occasions, and two years ago he expectorated a currant-jelly-like sputa more or less continuously for nearly three months, which induced me several times to seek carefully for signs of malignant disease, but without result; and the patient is now greatly better, all symptoms being in abeyance. My own experience would indeed lead me to say that hæmoptysis is not very uncommon in chronic basic pneumonia.

In the diagnosis of mediastinal growth from *aneurism* compressing the root of the lung, and setting up in it secondary inflammatory disease, we must have regard to the distinctions to be laid down between tumour and aneurism. Hæmoptysis would by no means necessarily decide the question in favour of tumour.

In endeavouring to come to a conclusion as to the *nature of the morbid growth* which we have ascertained to be present in the mediastinum, an inquiry so far as we at present know of no great practical importance, we may bear a few general facts in mind.

1. If the disease be primary in the mediastinum, it will be almost certainly lymphoma.

2. The younger the patient the more likely is it to be of this nature.

3. The presence of enlarged movable glands in the neck, or in other parts of the body (which may suppurate, but do not ulcerate) are also favourable to the diagnosis of lymphoma.

4. If the disease be secondary to a growth elsewhere it will be of the same nature as the primary disease, or allied to it within the range of pathological variation, *e.g.* If the disease make its appearance subsequently to a limb having been removed, or a joint resected for a malignant growth, we may feel confident that it is one of the sarcomata, either soft oval or spindle cell, or osteo-sarcoma, or enchondroma. If a fixed, nodulated, hard tumour were present in the neck or had been removed from the breast, we might expect the disease in the mediastinum to be cancerous.

5. So far as invasion of the lung goes, this feature is common to all these growths.

Prognosis.—The prognosis is unfortunately in all these present equally fatal. But we cannot say whether in the lymphoma some remedy may not hereafter be found to exerci control over the growth. The duration varies according to th involved by the tumour, it is rarely greater than a few month

Treatment.—There is no treatment to be adopted in these save to combat so far as is possible such symptoms as pain, ness, anæmia, &c. By attending to these points and to the di by local depletions when indicated, or in the case of comp with hydrothorax, by tapping, the lives of the unfortunate s from these dire maladies may be certainly prolonged and r more endurable.

THE DISEASES OF THE AORTA.

By R. Douglas Powell, M.D., F.R.C.P.

In the ensuing articles, the term "thoracic aorta" will be used inclusively as applying to that portion of the main arterial trunk which is contained within the thorax, having its origin at the left ventricle of the heart, and escaping through the diaphragm to become continuous with the abdominal aorta at the level of the last dorsal vertebra. In its passage from the ventricle to the left side of the third dorsal vertebra, the aorta describes a somewhat twisted curve, and this *arch* of the aorta is divided for convenience of anatomical description into an ascending, a transverse, and a descending portion. That portion of the vessel extending between the third dorsal vertebra and the diaphragm, to which the term thoracic aorta is sometimes exclusively applied, is better described as the descending thoracic aorta.

The function of the aorta—that of receiving and distributing, in more equable currents, and with the least possible conversion of motive force, the blood impelled into it with each systole of the left ventricle—is mainly performed by the arch, and for the most part mechanically, by virtue of its being a curved elastic tube furnished with appropriate valves. But the anatomical structure of its walls, the organic muscular fibre they contain, and the phenomena occasionally witnessed in disease, forbid our regarding the whole function of the aorta as being quite so simply discharged. We have organic muscular fibre nowhere else in the body save where the property of muscle is appreciably exercised, at least to that ill-defined extent which we characterise by the term *tonicity*. It is by the local deprivation of this unobtrusive form of muscular activity through nervous agency that we may most reasonably explain those violent aortic pulsations which are occasionally met with in nervous people; and no doubt mental depression and anxiety exert largely through this means what influence they have in predisposing to aortic aneurism.

AORTITIS.

Acute inflammation of the aorta, in the sense of an acute exudative inflammation, is a disease of very doubtful, if not impossible, occurrence. Professor Lebert,[1] after detailing the symptoms that have

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[1] Virchow's Handbuch der spec. Path., Krankheiten der Blut-und Lymph-gefässe, 1855.

been ascribed to the disease by Frank, Bizot, and others, co
that, in the course of twenty years' experience, he has not s
case, either clinically or anatomically, corresponding to it.
fessor Rindfleisch[1] observes that, "apart from thrombotic
and phlebitis, there is hardly such a thing as acute inflamma
the walls of the vessels," save, as he proceeds to explain, in s
their external coats, which must be regarded as part of the
connective tissue, may partake in any contiguous inflammation
even in this partial manner the aorta is very slow to share
processes, and when it does so the inflammation is very chro
limited, giving rise to no special symptoms.

On the other hand, it must not be denied that such a dis
acute inflammation may affect those portions of the walls of th
which are vascular in the usual way, and involve the non-v
inner coat in an irritative and disorderly cell proliferation, af
manner in which acute inflammation affects such tissues.
cases have not, however, yet been distinguished by definite
symptoms. The very striking cases of aortitis related by D
Sir Dominic) Corrigan, in the *Dublin Medical Journal* for 1838,
in the light of a newer pathology, be regarded as examples of at
of the vessel running a rather rapid course.[2] The case
most nearly perhaps of any on record presented the symptoms
have been ascribed to acute aortitis—viz., fever, rigors, tum
action of heart, with intense and painful throbbing of the aor
embolic infarction of distant organs—is that related by Mr. M
the "Medico-Chirurgical Transactions," vol. xlvii. p. 129, in wh
endeavoured to promote consolidation of an aortic aneurism th
rapidly making its way through the thoracic pareites, by the
tion into it of numerous coils of fine iron wire, but after dea
inflammation was found to be confined to a secondary sac
aneurism which had perforated the chest-wall. In a rema
case recorded by Dr. Parkes in the *Medical Times and*
February, 1850, there was *post-mortem* evidence of recent i
matory disease, affecting a large portion of the descending aorta,
however, appeared to have supervened upon disease of old st
which had doubtless much modified the anatomy of the walls
artery and the ultimate distribution of its nutritive vessels
disease in this case was not attended with any characteristic sym
during life, the absence of which might, however, have been
the almost insensible condition of the patient while under obser

It would be unprofitable to dwell further upon a disease
existence of which, as a primary affection, there is, we think
be said, as yet no sufficient clinical or *post-mortem* evidence.

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[1] Pathological Histology, vol. i. p. 249, Dr. Baxter's translation for Sy
Society, 1872.

[2] The cases described by Norman Chevers, Guy's Hosp. Rep., 1841, are so dif
recognise in accordance with the pathology of the present day, and so complicat
other diseases, as to be useless for clinical illustration.

AORTIC ENDARTERITIS, ATHEROMA.

The inflammatory process by which the aorta is commonly affected is necessarily of a slower kind from its attacking primarily a non-vascular tissue—the internal coat of the vessel. It consists essentially of proliferation of the cell elements of this coat, commencing in its deeper layers and extending sooner or later to the middle and external tissues.

The pathology of this process is minutely described in another article; its effects upon the walls of the aorta—which are spoken of collectively as atheroma—may be referred to in three stages or degrees—(1), patchy thickening, with some softening, mainly affecting the inner coat, unevenness of the inner surface, and diminished elasticity of the vessel; (2), fatty degeneration of the affected tissues, fibroid thickening of the whole vessel wall; (3), crumbling down, or infiltration with calcareous salts, of the degenerated internal or middle coat; great consequent roughening of the inner surface of the aorta, and increased brittleness of the vessel.

In the earlier stages of aortic endarteritis there is sometimes narrowing of the calibre of the vessel from the intrusion of its thickened walls; but dilatation almost always takes place later on in the disease. The later stages of atheroma, softening, and calcification, are little more than the effects of imbibition and chemical change acting upon a part which has lost its vitality.

Atheroma may then be defined as degeneration of the coats of the aorta, the result most commonly of preceding inflammatory change (endarteritis), but, it must be added, sometimes occurring primarily as fatty transformation from senile decay.

The *seat* of the disease is most commonly at the commencement of the aorta, and this is the portion most affected even in those cases in which other parts of the vessel are involved. Moreover, we can rarely obtain clinical evidence of atheroma affecting the aorta beyond its ascending or transverse portion.

ÆTIOLOGY.—It is of some importance clinically to bear in mind respecting the pathology of atheroma, with which we are now concerned, that the slow inflammatory changes which lead to its production are, in the majority of instances, of a degenerative kind from the first—that is to say, they are associated with some constitutional cachexia or with senility. Even in those cases in which the disease occurs in earlier life, and apparently as the result simply of undue arterial strain, there may usually be strongly suspected some antecedent impairment of nutrition to account for that sensitiveness or want of resilience to strain which leads to the setting up of atheroma.

It has, however, been shown very clearly by the clinical observations of Dr. Clifford Allbutt and Mr. Myers, and the patholcgical inquiries of Dr. Moxon and others, that aortic atheroma is particularly common among those who are engaged in occupations of a constantly laborious kind—strikers, bargemen, those who work heavy pumps,

&c. ; and Dr. Allbutt regards daily continued heavy labour a more efficacious in producing this result than intermittent even a more severe kind, such as the athletic sports of the classes; but he also lays stress upon his opinion that de circumstances of life, bad air and food, greatly favour atheroma from strain.[1]

Rheumatism, gout, syphilis, and kidney diseases are the n most predisposing to atheroma. Intemperance and he tendency hasten its appearance. Of senile changes, atheroma of the most constant, but it only rarely comes under our observation, from the diminishing activity and vigour of the tion with advancing age rendering the results of atheromatous in the aorta less likely to manifest themselves.

Mr. Francis H. Welch of Netley has in a recent paper rea the Medico-Chirurgical Society of London (1876) contended t dular disease of the aorta (endarteritis) is one of the most f internal lesions of the syphilitic virus and also one of the produced—a view supported by some high military authoritie Welch found that out of 56 cases of syphilis terminating fatal specific lesions 60·7 per cent. showed aortic nodulations. Agai cases dead of aortic aneurism 50 per cent. at least were s infected with syphilis. Mr. Welch's important paper did n without criticism, and it will no doubt do much to hasten the s of a very complex question as to the real potency of syp developing atheroma. The main issue rests upon what is rega evidence of syphilis both during life and after death.

SYMPTOMS AND PHYSICAL SIGNS.—The symptoms of athero change in the aorta are always obscure; and extensive disea exist without any symptoms being complained of; nor on th minutely careful physical examination can we in all cases asse there is or is not atheromatous disease of the main vessel The secondary results of atheroma are, in the first place, mecha viz., dilatation, aneurism or rupture of the vessel, or embolis the conveyance of masses of fibrine which have been entan the roughened surfaces to distant parts; and it is only whe secondary phenomena begin to arise that symptoms or si disease present themselves. Our object must then be to di at the earliest possible moment, the commencing secondary quences of atheroma, so as to be on our guard, so far as is p against their further extension.

Attacks of angina or of palpitation, occurring independe effort, but readily brought on by exertion, are suggestive form of the disease; but it as often happens that some casual tom which, so far as symptoms are yet classified, might mea thing or "dyspepsia," in the man before us directs our att to the heart. The patient is of an age at or beyond that of life, which is in favour of the probability that such cardiac attacks may be dependent upon dyspepsia (or, in the case of women, climacteric hysteria), and the presence or absence of these conditions must of course be carefully ascertained; but their existence must not be regarded as sufficient to exclude the graver malady, for they frequently coexist with and complicate aortic disease. There is nothing characteristic in the appearance of the patient; he may be thin and cachectic looking, or the reverse; but whether his appearance suggests such questions or not, rheumatism, gout, syphilis, and intemperance should be inquired for, and the urine repeatedly examined for albumen or morbid deposits. On examination, the radial and brachial arteries will commonly be found more rigid and inelastic than natural.

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[1] On Overwork and Strain of the Heart, and Aorta.

Although during an attack of dyspnœa the heart's action is tumultuous and the pulse alarmingly irregular, yet at the time the patient comes under observation the cardiac movements may be quite steady, with perhaps an occasional intermission, there is some evidence of hypertrophy of the left ventricle (increased impulse with muffled sound), and perhaps nothing else can be discovered; from these patients being big-chested and more or less emphysematous, it is also sometimes difficult to judge of the cardiac hypertrophy. In more marked cases, however, there is with indistinctness of the first sound, accentuation of the second and a *short systolic, or rather post systolic, murmur* over the aorta beyond the valves. It may be that this murmur is only discoverable when the heart is acting strongly as from excitement, or after taking a few turns up and down the room—and in suspicious cases this exercise prior to a second auscultation should never be omitted; the murmur does not displace the first sound, but is superadded to and immediately follows it. In other cases there is a *partial* replacement of the second sound by a fine diastolic murmur. These latter physical signs are often not to be found for the first few months, during which the patient has presented suspicious symptoms, and *their supervention in this way is the most significant feature in the history of such a case for diagnosis.* In the upper sternal region both the cardiac sounds are accentuated, and may be attended even with slight shock *to the ear;* in order correctly to value this sign, however, a reverse precaution to that mentioned above should be adopted—we should note that the patient be quite calm, and if possible, make a second examination after he has been lying down for a short time.

At a later period, signs of decided dilatation may become apparent, there may be some dulness ever the aortic region, and some pulsation—rather flapping than thrusting—may be felt by the finger at the second interspace close to the sternum. The bruit becomes more distinct, sometimes very rough and accompanied by fremitus, when calcareous degeneration may be presumed to be present. The dyspnœa increases, and the anginal attacks may become more frequent and severe; or, on the other hand, they may disappear; but palpitation is

always more persistent. The symptoms of embolism may no
on, among which hemiplegia, rigors and hæmaturia, superficial
rhages, and gangrene may be enumerated; or sudden pain, dy
and faintness may announce the commencement of a sac
aneurism, or death may suddenly take place from cardiac sync
rupture of the aorta. In other cases sacculated aneuris
imperceptibly arise.

The acute symptoms signalising the formation of a dis
aneurism may be the first to announce to us the existence
preceding atheroma, and it is unnecessary to say that the dise
phenomena of which are thus related in chronological orde
first present itself to our notice at any of the stages referred
all cases of suspicion the general state of the circulation sh
carefully examined, the aid of the sphygmograph being sought.

DURATION.—The duration of aortic endarteritis or atheroma
be precisely stated, from the insidious manner in which the
commences. It may be considerably, perhaps indefinitely, pro
by careful management, hence the importance of its early recog
The disease proceeds to its fatal termination either by simple p
leading to rupture of the vessel, or by embolism of the brain o
organs from conveyance of debris or fibrinous plugs; or by dil
of the aorta, or direct involvement of its valves, giving rise to
petency with its attendant cardiac results, or, finally, by the for
of a sacculated or dissecting aneurism. In calculating the *pr*
the present duration of the disease and the progress it has
made in one or other of these directions has to be considered.

TREATMENT.—The treatment of aortic degeneration is purely
tive. A careful regulation of the diet so as to avoid both overl
of stomach and too long fasting, is of the first importance; stim
should be reduced to a minimum, or dispensed with altogether, a
hepatic function should be carefully attended to. A mild but
climate with level walks and carriage exercise are desirable. O
the aromatic stimulants and antispasmodics are most useful
the attacks of dyspnœa. The subcutaneous injection of mor
very useful in warding off the attacks, when, as is not infreq
the case, they show any tendency to recur at definite times.

ANEURISM OF THE THORACIC AORTA.

BY R. DOUGLAS POWELL, M.D., F.R.C.P.

ANEURISM AT THE SINUSES OF THE AORTA.

NEURISM affecting the very commencement of the aorta, either at or mmediately above one of the sinuses of Valsalva, rarely attains a suffiient size to assume special characters of its own before death takes lace, either from its rupture into the heart or pericardium, or indirectly rom the grave derangement of the valves and orifices at the base of he heart it has occasioned. It may therefore be more conveniently onsidered here than among the larger aneurisms affecting the rest of he aorta. The aneurism occurs usually above the right coronary valve, ext most frequently above the intercoronary valve or between these wo (Sibson). It is also always of the sacculated variety. The pouch ecessarily projects in most cases into the right side of the heart at or ear the commencement of the pulmonary artery.

SYMPTOMS AND PHYSICAL SIGNS.—In a certain number of cases there re no symptoms to attract our notice to the heart, until the patient uddenly dies from rupture of the sac. In other cases the symptoms nd signs are those of atheroma, with some dilatation of the first ortion of the aorta. The expansion of the portion of the aorta orming the base of the aneurism, tends to displace downwards the ttachment of the aortic valves, and if situated above the junction of wo of these valves necessarily occasions great incompetency, with all he signs and symptoms of aortic regurgitation. The pouch by proecting as it most commonly does, towards the base of the pulmonary rtery, tends also to displace its valves or narrow its orifice, and hence here may be a systolic or a diastolic murmur situated in the pulnonary region. This latter sign in particular might lead us to uspect the disease. Hypertrophy of the left ventricle, with or without similar affection of the right, is usually present. In two cases vhich have come under my own observation, the hypertrophy and ilatation of the right side of the heart were very marked.

DIAGNOSIS.—The *diagnosis* can rarely be made with certainty. It s almost impossible to single out from among the symptoms which nay be accounted for by the many attendant lesions, those peculiar o an aneurism rarely exceeding the size of a filbert, and buried in

the base of the heart. Yet, where we have evidence of these le
of aortic atheroma with some dilatation, and of regurgitation t
the valves—the existence of aneurism should always be re
upon as possible; and if there be any murmur detected o
pulmonary artery, accompanied by marked hypertrophy and
tion of the right side of the heart, the presence of aneurism
fairly assumed.

The termination of the disease is usually by rupture in
pericardium or right side of the heart. Death may ensue, h
from the valvular derangement occasioned by it, or in some ot
from the extensive disease of the vessel by which it is most co
accompanied.

The general treatment of this disease is identical with
atheroma.

Aneurism of the Thoracic Aorta beyond the Valv

Aneurism of the aorta is a preternatural dilatation of that
at some portion of its course. The dilatation may be *general*, i
ing the whole circumference of the vessel for a certain lengt
such an aneurism may assume the *fusiform*, *cylindrical*, or *g*
shape. The aneurismal expansion of the vessel is more com
partial, from the yielding before the blood-pressure of some c
scribed portion of the arterial wall previously weakened by d
which bulges outwards from the vessel as a bud, or *cul de*
gradually increasing dimensions. This variety is spoken of
sacculated aneurism; it is always associated, however, with
general enlargement of the arterial channel.

These varieties of aneurism are more easily classified in the m
than distinguished clinically. Among the *circumscribed* ane
of which the sacculated variety is the type and form of most fr
occurrence, we must also include, for purposes of description, the
lar[1] aneurism, and those aneurisms strictly speaking perhaps
dissecting kind, *i.e.*, commencing suddenly with rupture of the in
coat, but in which the lesion is limited, and the further progress
disease is by gradual expansion. No doubt more aneurisms, reg
even *post mortem* as sacculated, originate in this way than is gen
supposed. The ordinary *fusiform aneurism* is in its slighter d
more commonly spoken of under the simpler name dilatation
aorta, and has already been referred to in speaking of atherom

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[1] This is perhaps the best term to apply to those cases in which the dilatation a
the whole circumference involves only a comparatively limited portion of the le
the aorta. It is not a new one, being used by Dr. Walshe in the same sense, tho
cases referred to he regards as of extreme rarity. An aneurism of this globul
presents all the physical signs and clinical phenomena of a sacculated aneurism,
perforation of the thoracic parietes, as in Dr. Murchison's case related at p
One or two instances of this variety, which seems to be more frequent in the desc
thoracic aorta, have lately appeared before the Pathological Society.

usually affects the first portion of the aorta, and, when extensive, yields many of the signs of aneurism presently to be described.

ÆTIOLOGY.—Speaking generally, whatever increases the pressure of blood within the aorta or impairs the resisting power of that vessel, favours the production of aneurism. Increased propelling power of heart and increased resistance to the escape of blood from the aorta, into the vessels beyond, are the two conditions which, separately or combined, augment the pressure of blood within the vessel: disease of the walls of the vessel, of whatever kind, diminishes its power of resisting the normal or enhanced blood-pressure.

In discussing more minutely the ætiology of aneurisms of the aorta, we must refer separately to the disease as occurring, as a result of senile changes, and as being prematurely produced by artificial or accidental circumstances. Senile decay of the arterial wall is one of the natural causes of aneurism. In the normal progress of age, degeneration commences in the large vessels, and probably too in the smaller ones, before the tissue of the heart suffers in nutrition;[1] indeed, the first effect of this arterial decay is, as is well known, a certain increase in the muscular power of the left ventricle to compensate for the increased resistance to the circulation through the more rigid vessels; and it is at this period of advanced middle life that aneurism is most frequently met with—but sometimes age in these special tissues is hurried on by favouring disease. Beyond the period of advanced middle life the tendency to sacculated aneurism is lost, the bulk of the blood is diminished, the nutrition of the heart begins to suffer and the vigour of the circulation becomes correspondingly lessened; a lower activity of life is thus necessitated which is quite normal to advancing age and in harmony with the changes taking place in the tissues. In old age, however, it sometimes happens that the senile atheromatous change—no doubt aided by some attendant (secondary ?) inflammation—proceeds to actual softening and erosion of the inner tunic of the aorta, and the blood then insinuates between the coats of the vessel, and a dissecting aneurism arises.

Sub-acute and chronic arteritis, the varying stages and modifications of which have already been referred to as accounting for the production of most of the appearances recognised under the term atheroma—thickenings, scars, erosions, calcifications, &c.—is the diseased condition most commonly preceding aneurism; indeed, it is through the intervention of arteritis that all the known causes of aneurism of the aorta, and, perhaps, of other vessels, become effective. The constitutional states, then, which tend to produce atheroma, also favour the occurrence of aneurism—hereditary predisposition, rheumatism, gout, syphilis, kidney disease, alcoholism. Even strain, with but rare and violent exceptions, leads to the production of aneurism, not simply by rupturing the coats of the aorta, previously healthy, but by overstretchin violently exercising them, and thus setting up local athero disease of the slow inflammatory type. Dr. Moxon[1] has together so many arguments to prove the direct effect of st producing atheroma as to have, I think, settled the questio shows how the disease affects first the aorta in the region valves and at its ascending portion, and that the inflammatory ration occurs in points arranged longitudinally in the course vessel. Thus arising, it is not difficult to understand how any little disease "rifts" may slowly widen to the production of aneurism, or may, under some severe effort, suddenly giv with those acute symptoms which occasionally usher in the disease. Hereditary predisposition has not been found to exis in exceptional cases, in aneurism. One remarkable case came my own observation seven or eight years ago, in which there tolerably trustworthy history of a mother and four sons dying ternal aneurism. Dr. Fuller[2] relates the case of a gentleman paternal grandfather, uncle, and father, had all died from an and whose sister was labouring under that disease.

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[1] This statement, it must be admitted, is based rather upon general clinical experience than upon any exact inquiry into the natural relative progress of age in the different tissues, which is as yet wanting in medical literature. It is, I believe, however, in accordance with the impression of most physicians, and is in all probability exactly true.

Although rheumatism is usually enumerated among the d predisposing to aneurism, it has hitherto been included amon general causes as syphilis and renal disease, on the tacit standing that the "rheumatic diathesis" is favourable to the occ of premature arterial decay. Whether this be the case is a I think, a question for further inquiry; but rheumatism mus more definite sense be regarded as a possible cause of aneuris may judge from the history of some cases which have come un own observation.[3] It has been found in certain cases of rhe fever with aortic valve disease, that the aorta beyond the valv presented patches of arteritis corresponding with the imping against it of vegetations fringing the margins of the valves. the rheumatic endocarditis ever extend beyond the valves to p endarteritis affecting the aorta at its commencement? I hav seen *clinical* evidence in one case which would lead me to this question in the affirmative. The case was that of a lad a in whom aortic aneurism occurred traceable to two attacks of matism occurring at the ages of eleven and twelve. No other for the aneurism could be made out, and the patient was too for degeneration of the vessel, save of an acute kind, such as possibly be occasioned by rheumatic fever.

The operation of aortic regurgitant disease of the heart, left by rheumatic endocarditis, as a cause of aneurism, may be conceived in persons at or beyond middle life. I have met wit examples of aneurism, the histories of which will, I think, admit of no other interpretation; and in two other cases I have had reason to suspect the disease to have arisen in this way. Greatly increased power of cardiac systole is required in aortic regurgitant disease to carry on the circulation with an aorta whose action is crippled through imperfection of its valves: the whole shock of this extra power is received of course by the ascending portion of the aorta. Moreover, instead of the unduly forcible impulse of blood being received by an aorta already containing a certain residuum of blood sufficient to diminish the shock, the great vessel is, on the contrary, more empty and flaccid than natural at the moment of ventricular systole, the blood in regurgitant disease escaping back into the ventricle. The effect of this increased impulse, or shock, upon the aorta must be to predispose it to the occurrence of atheromatous disease and of subsequent expansion.

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[1] Guy's Hospital Reports, series iii. vol. xvi. p. 448; also recent work on Path Anatomy by Wilks and Moxon, chap. "Aneurism.

[2] Diseases of the Chest, p. 656.

[3] *Vide* Clinical Transactions for 1874.

Intemperance, mental emotion, and violent exercise operate as causes of aneurism by increasing the blood pressure within the aorta. Dr. Rendle's statistics of cases of internal aneurism occurring among the female convicts in the Queen's Prison, Brixton, conclusively show the effect of mental depression and excitement in predisposing to aneurism. As already hinted, loss of muscular tone of the vessel, through nervous influence,[1] may take a more important part in such cases in the origin of aneurism than is at present allowed. Mechanical impediment to the circulation through the great vessels beyond the aorta is, however, perhaps the most important cause of aneurism, with special reference to which the malady will be found to prevail more at certain ages, among those following certain occupations, and, with some reservations, in the male sex.

In an exhaustive essay on the ætiology of diseases of the heart among soldiers, Mr. Myers[2] gives the statistics of Inspector-General Lawson ("Army Medical Report," 1866), showing that the *deaths* from aortic aneurism are in the army eleven times greater than among the civil population; and he accounts for this enormous disproportion by the tightness of the dress and accoutrements of the soldier occasioning greatly increased blood-pressure within the aorta during any violent exercise, by compressing the great vessels of the neck and upper extremities. In proof of this Mr. Myers gives one set of 703 cases of aortic aneurism from Dr. Sibson, in 420 of which (59·7 per cent.) some portion of the ascending aorta was involved. He also gives—in support of Dr. Sibson's view that that portion of the aorta on which there is most strain is most often affected with aneurism—109 other cases, culled from the Netley Hospital records, and from those of his own regiment, in 75 (about 70 per cent.) of which

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[1] Niemeyer refers to palsy of the vasomotor nerves as a questionable cause of aneurism, though he gives Rokitansky's opinion in favour of it.

[2] On the Ætiology and Prevalence of Diseases of the Heart among Soldiers. London, 1870.

the disease affected the ascending portion or arch of the vess
thus sucessfully endeavours to locate the cause of the dis
soldiers from its point of manifestation being disproportionatel
frequent in them at that portion of the aorta which is at or ab
origin of the great vessels of the neck and upper extremitie
further evidence of the effect of strain in producing heart
among soldiers, Mr. Myers mentions the significant fact that
them aortic valve disease is twice as frequent as mitral, whe
civil life mitral disease is slightly the more frequent. He als
to the very large proportion of valvular heart diseases in th
which are not traceable to either rheumatism or albuminuria,
diseases answerable for most such cases occurring in civil life.
of course, be remembered that wherever the obstruction may
aorta at its origin must be most importantly affected; for it
next to the ventricle, that the supplementary force is gath
overcome it, and perhaps the great and almost sudden cha
the mode of life on entering the military ranks—from that of th
moving, slouching, ill-fed farmer's lad, or the loafer from am
unhealthy recesses of large towns, to that of the smart, straight
eating and more or less plethoric soldier—should be also tak
account, as well as his disadvantages in dress, in consideri
effects of the soldier's occupation in predisposing to aneurism.[2]
ever this may be, it will be readily perceived that arterial
tending through the medium of atheroma to produce an
among other diseases of the heart and aorta, is the common
of many of the conditions of the soldier's present life and tr
Sailors, with an equal liability to great physical effort, and
in about equal proportion with the taint of syphilis, although
subject to aneurism than civilians, are less so than soldiers
greater amenity in this respect appearing due mainly to their
dress and more gradual training. The occupations of hamm
lightermen, smiths, and others, necessitating long-continued mu
effort, predispose to aneurism.

AGE.—Aneurism of the aorta may occur at any age; it is, ho
extremely rare before twenty, and is most prevalent between the
forty and fifty. A small number of cases, 5 or 6 per cent., occur
thirty. Professor Lebert[3] finds the disease most prevalent betwe
ages of forty-five and sixty. In Dr. Crisp's[4] tables, 132 out of 17

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[1] Taking the whole of Dr. Sibson's 880 cases, of which in 460, or 52·3 per ce
disease was situate at either the ascending or transverse aorta, or between the t
further making allowance for the fact that these cases were of all ages, where
quoted by Mr. Myers were within the age of effective service, the preponderanc
affection in ascending aorta among soldiers appears still more considerable.

[2] It is a fact worthy of note that severe gymnastic exercises have only been in v
the army within comparatively recent years. I have, however, met with aneu
several soldiers who have never been subjected to them.

[3] Virchow's Handb. der spec. Path., Bd. v. abth. ii.; Krankheiten der B
Lymph-gefässe, p. 25.

[4] Diseases of the Blood-vessels, p. 135.

occur between the ages of thirty and sixty, this wide margin being, as before explained, accounted for by certain habits and constitutional states anticipating by disease the effects of age upon the vessels. The great majority of aneurisms occurring between these periods of life are of the circumscribed sacculated or fusiform kind; in advanced life, on the other hand, those cases which do occur are most commonly of the dissecting kind (Peacock [1]).

SEX.—Males are much more liable to true aneurism of the aorta than females, though the latter are by no means exempt from the disease. Of circumscribed aneurism of the aorta from two-thirds to four-fifths of the cases occur in males (Crisp, Peacock, Blakiston); on the other hand, dissecting aneurism appears to be of as frequent occurrence in women as in men. That this disproportion between the sexes is due entirely to the difference in their habits of life is apparent from the statistics of Dr. Rendle, already referred to. Mr. Holmes [2] remarks that "internal aneurisms seem equally if not more common among women when their way of life exposes them to the vascular excitement consequent on intemperance, vice, and mental emotions."

Though aneurism appears to prevail more in Great Britain than in other countries, this excess is attributable to our rougher mode of life rather than to any climatic influence.

SYMPTOMATIC HISTORY AND SYMPTOMATOLOGY.—As a rule the commencement of aneurism of the aorta is unmarked by symptoms, and the disease may continue latent up to the time of death. The so-called *exciting causes* of aneurism, those acts or accidents which seem immediately to determine the commencement of the disease, cannot well be classified; when recognisable they consist either of some temporary exaggeration of usual daily toil, or of some shock, or fall, or blow, violently affecting the circulation. Such causes are, however, but rarely sufficient to account for *internal* aneurism if we attempt to isolate them from the predisposing, *i.e.* the true causes of the disease. In the sacculated or circumscribed forms the history, when any can be discovered, may be referred to one of the following types. In one case there may long have been signs of failing health and nutrition, and positive signs of degenerative disease of the aorta may have previously been detected. In another the patient has perhaps enjoyed robust health and indulged in active pursuits and free living until, after a fall from a horse, or a blow, or a sudden violent effort, he feels a momentary pain in the chest and faintness, soon passing out of memory but recalled by the consciousness, on subsequently assuming his wonted exercises, of a gradually increasing shortness of breath and palpitation, with deep-seated pain in the chest: the dyspnœa becomes habitual, and he is ever conscious of too great a pulsation within the chest which

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[1] On Dissecting Aneurism. Edin. Surg. and Med. Journal, vol. lx. p. 291, 1843.
[2] System of Surgery, vol. iii. p. 419.

disturbs his rest. He then seeks advice. Sometimes the sy
come on more acutely—*e.g.* a tailor on lifting while in the "sq
posture the heavy seam press he has been accustomed to wield
in the same way, is seized with agonising pain in the left bre
longed faintness and violent palpitation, and soon a pulsating
appears in the aortic region, where he had felt "something gi
The symptoms in other cases come on almost imperceptib
cough, dyspnœa, and partial aphonia, attributed to cold, yet th
will, on being questioned, perhaps date them back to some pr
labour or "heavy job," as in the case of a blacksmith or lighte

At the time of applying for advice the symptoms vary in thei
and intensity with the seat of the tumour and the direction i
it is growing. They are all due to pressure upon the neighbouri
causing their displacement or erosion, interference with the
of air and food tubes, irritation or destruction of nerves, and tl
be thus enumerated nearly in the order of their frequency
dyspnœa, cardiac or pulmonary, or both, persistent in greater
degree, but often associated with paroxysmal attacks; *voice*
husky, uncertain, or whispering; *cough* dry, hoarse, or ringing
geal, *stridulous breathing, headache, disordered vision,* loss of p
positive *paralysis* of lower extremities.

Few if any of the symptoms as thus enumerated can be reg
specially distinguishing aneurism from other tumours in th
their significance rests rather upon their grouping and upon
characters about them which require further consideration.
istence of an aneurism may sometimes be inferred from the p
of certain symptoms whilst the physical signs are as yet most
and otherwise insignificant.

The *pain* in aneurism is of a wearing, aching, or burning ch
corresponding in position with the seat of the tumour. It is
pain, but associated with paroxysmal pains of an evidently n
character, radiating in the course of contiguous nerves. The fir
of the disease is occasionally ushered in, as before said, by ver
suffering.

The *dyspnœa* present in cases of thoracic aneurism is of
kinds and grades, and has many causes. (1). The mechanism
circulation being disordered, any effort giving rise to an extra
upon the heart occasions dyspnœa. (2). The simple fact of the
a tumour within the chest compressing and displacing its oth
tents necessitates some degree of dyspnœa. The fact of the
being pulsatile and of dimensions varying slightly from time t
with the fulness of the circulation may serve to give a disting
feature to the dyspnœa, viz., its more marked sensitiveness to con
tending to disorder circulation than in the case of other tumou
certain deep-seated throbbing is sometimes complained of afte
or excitement. But (3), the dyspnœa most characteristic of an
occurs in paroxysmal attacks, in one of which the patient not
quently dies. Much dispute has arisen as to the mechanism

form of dyspnœa, and upon the views we hold respecting it will depend our treatment of cases of impending suffocation from this cause.

The pneumogastric nerve is sometimes affected in the disease; it may be either compressed and flattened by the tumour, or destroyed and incorporated with the sac by inflammatory change. More commonly one or both of the recurrent branches are affected, the left most frequently so by direct compression, or the right indirectly by dragging of the tumour upon the origin of the subclavian. Either paralysis or irregular spasm of the laryngeal muscles on one or both sides may thus be occasioned. Among those who attribute these attacks of dyspnœa to disturbance of the innervation of the glottis, some regard them as due to *spasmodic* closure of the glottis, others to *paralytic* closure. Spasm of the glottis is the cause to which the dyspnœa is most commonly attributed.

It is generally held that, to quote the words of Dr. John Reid,[1] "all the muscles which move the arytenoid cartilages receive their motor filaments from the inferior or recurrent laryngeal nerves. And as the force of the muscles which shut the larynx preponderates over that of those which dilate it, so the arytenoid cartilages are carried inwards when all the filaments of one or both nerves are irritated." This is the view upon which those who hold the spasm theory mainly rely. M. Krishaber[2] observes, as the result of some experimental inquiry, that in paralysis of the chords either from destruction of the recurrent nerves or division with the knife of one or both of them, there is no dyspnœa, the glottis being actually more patent under such circumstances than natural. This view, however, is entirely opposed to the conclusions drawn from similar experiments by others. The experiments of Legallois and John Reid distinctly show that on section of the recurrent nerves the paralysed chords are sucked together with each inspiratory effort, giving rise to suffocation or great dyspnœa. Some experiments I have myself made on the cat entirely confirm in this respect those of Legallois[3] and Reid, while in another particular, the effect of irritation of these nerves upon the larynx, they, so far as they have gone, lead me to hesitate in accepting the conclusions of these and other experimenters. For while the cat was in a state of urgent distress from the sucking together of the paralysed chords with each inspiration, first one inferior laryngeal nerve, then the other, and then both, were galvanised with a weak current, and in each case the corresponding arytenoid cartilage was powerfully rotated outwards, so as to widely open the glottis and instantly relieve the dyspnœa.[4] While, then, I think that further experimental inquiry will tend to show th modic closure of the larynx may not in man be caused by i of the recurrent nerves, clinical observation is also in favou dyspnœa of aneurism, so far as it is laryngeal, being due to sis. When the larynx is at all affected, paralysis—denote altered voice, and observable by the laryngoscope — is th usually present. Dr. Habershon[1] has found actual atroph laryngeal muscles on the affected side. Dr. George John Dr. Baümler in the 23rd and 24th volumes of the Pathologica actions respectively, have each recorded a case in which paraylsis of the chords was found during life, and *post mor* laryngeal muscles on both sides were found to have undergo phic changes. In these two cases the trunk of the vagus, as the recurrent nerve on one side, was found compressed tumour, the nerves on the other side being free. Dr. considers that the bilateral paralysis in these cases may ha due to the compression directly paralysing the muscles same side through the recurrent, and causing reflex paralysi muscles on the opposite side through the trunk of the p gastric and the efferent nerves in relation with its centr Johnson holds that bilateral spasm may be occasioned in a way.[2]

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[1] Physiological Researches, No. iv. Experimental investigations into the functions of the eighth pair of nerves, 1848. Dr. Sanderson (Handbook of Practical Physiology, p. 298) regards all the intrinsic muscles of the larynx as expiratory, the widening during the inspiration being a condition of general relaxation.

[2] Comptes Rendus des Séances de la Société de Biologie, October 1866, d' l'Opportunité de la Trachiotomie dans les Anévrismes de la Crosse de l'Aorte.

[3] Sur le Principe de la Vie, 1812.

[4] These experiments were made in the summer of 1874 with the kind help of Dr. M. Bruce and Mr. Schäfer.

But how does paralysis of the chords give rise to occasional pa of dyspnœa? The mechanism of these attacks of dyspnœa s be either (*a*) that owing to sudden enlargement of the aneuris excitement of heart, after a full meal, or from mental emotion or exertion, the increased pressure on the nerve renders a partial p complete, and a paroxysm of dyspnœa occurs from sucking t of the flaccid chords; or, (*b*) as one may observe in some c which the chords have been destroyed by ulcerative disease, s they are paralysed, effectual cough being impossible, mucus at the glottis and gives rise to dyspnœa. And moreover w remember that—as may be well seen in experimenting with —during any excitement of breathing, the dyspnœa already from paralysis of the chords necessarily becomes more urge more powerful the inspirations the more completely is the glotti by atmospheric pressure. But there are at least two other ca paroxysmal dyspnœa in aneurism, affecting the trachea or Dr. Bristowe, in a valuable communication to the St. Thomas's H Reports for 1872, expresses his belief that the dyspnœa of intrat tumour is only rarely purely laryngeal, and contends that it i commonly due to direct narrowing of the trachea by the pres the aneurismal or other tumour, and to accumulation of mucus point of stricture, acting merely mechanically by plugging th rowed opening, and perhaps causing in addition some spasmodi traction of the tube. He illustrates his view by several case there are many others, one of which has lately come under my own observation, which bear no other interpretation; and I think his explanation will be found applicable to those cases in particular in which there is marked stridor on one or both sides of the chest, according as the trachea or one main bronchus is pressed upon. In such cases of course all idea of relief by tracheotomy must be abandoned. Again in some cases of aneurism, particularly when affecting the third portion of the arch, paroxysms of dyspnœa, closely resembling those of asthma, may be occasioned by compression of the small branches of the pneumogastric forming one of the pulmonary plexuses. (Gairdner.) Direct pressure upon the trachea may also, however, as I have seen in one remarkable instance, give rise to dyspnœa having very closely the characters of asthma.

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[1] Medico-Chirurgical Transactions, vol. xlvii.

[2] Vide Medico-Chirurgical Transactions, for 1875.

Palpitation, or cardiac dyspnœa, is usually an intermitting rather than a constant symptom in aneurism; it is generally complained of on slight exertion. Attacks of true angina pectoris are sometimes witnessed in cases of aneurism affecting the first portion of the aorta, and probably arise from pressure on the cardiac plexuses (Gairdner). There is usually more functional disturbance of the heart when the disease is thus situated. Acceleration of pulse is sometimes, however, a persistent symptom: in the case of a woman for many months under my notice with aneurism of the first portion of the arch, the pulse was constantly beating at a rate of between 130 and 140 per minute, the patient complained of palpitation, and assured me that the heart's action was not quickened from excitement at the times of my repeated observations.

Dysphagia, although often present, is a less constant symptom in aneurism than in other tumours in the same situation.[1] I have seen two or three instances in which an aneurismal tumour has caused a circular perforation of the wall of the œsophagus, without any distress having been complained of from difficulty of swallowing.[2] There is sometimes a spasmodic character in the dysphagia, which is regarded as of reflex origin.

Headache and *disordered vision* are occasional symptoms, the former referable to obstructed return of blood from the head, the latter to pressure upon the sympathetic affecting the size of the pupil.

Hæmoptysis, though it may occasionally, and to a slight extent, occur in the course of aneurism, from bronchial congestion or lung irritation, as a rule only presents itself as the final symptom in those cases in which rupture of the aneurism takes place into the trachea, œsophagus, or one of the great bronchi. Sometimes this final gush is preceded for a few days by a sanguineous tinging of

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[1] M. Leudet observes that dysphagia in compression of the œsophagus by aneurism of the aorta is often absent. "Recherche sur les Lésions de l'Œsophagie causés par les Anévrismes de l'Aorte." Compte Rendus de la Soc. de Biol., 3me sér. 1863, p. 180.

[2] For a good example of this, *vide* case of aneurism by Dr. Quain, Path. Trans., vol. xvii. p. 110. Dr. Fuller also refers to the occasional occurrence of severe lesion of the œsophagus from aneurismal pressure without any corresponding dysphagia, Diseases of Chest, 1862.

the scanty expectoration, and Dr. Gairdner mentions a case i
this expectoration preceded death by a considerable interv
Blakiston[1] has recorded a case in which fragments of dis
coagula were expectorated with blood two or three week
death, which materially aided the diagnosis of an aneurism
descending arch communicating with the left bronchus.

PHYSICAL SIGNS.—It is by the physical examination of the
before us—the observation of all the signs discoverable by
the hand, the ear, aided by the stethoscope, the laryngoscope,
sphygmograph—that we clinch the diagnosis as to the exist
probable seat of the aneurism which the symptoms present
us to look for.

The physical signs of aortic aneurism vary greatly in
tinctness with which they are manifested according to the
and size of the dilatation. It is, practically speaking, true
aneurism may be present and give rise to death by rupture
having ever presented any distinctive signs. Such signs,
even in obscure cases, are more often overlooked than abs
may usually be discovered on diligent examination. We
enumerate all those that may be found in tolerably obvi
typical cases.

*Summary of the Physical Signs that may be Observed in A
of the Ascending or Transverse Aorta.*[2]—It is in a considera
portion of cases at once evident on *inspection* of the patient
to the waist, that he is suffering from aneurism of the aor
observe the veins large, full, and tortuous in the humoral re
neck on one side, more rarely on both, a certain fulness, and
ing of the antero-posterior diameter of the upper chest
sternum on that side, or the superior portion of the sternum
rounded and prominent, marbled with blue veins. At th
prominent portion of the costal or sternal bulging, there
tinctly visible pulsation, or there may be a more confu
rhythmic shock apparent with each beat of the heart. So
the pupil on one side corresponding with the pulsating tu
notably smaller than the other.

On now employing *palpation*, one hand being applied to
of the apex beat, which is usually shifted a little downwards an
left, and the other placed on the tumour, we feel two centres o
tion, synchronous or nearly so, within the chest—a very signific
of aneurism. More carefully noting the character of the mo
pulse, we may observe it to be distinctly heaving, expansile, sp

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[1] Diseases of the Heart, p. 56, case 24.

[2] These aneurisms are specially referred to here, not only because they are
common, but because of them alone can any general description includin
essential phenomena of the disease be given. Aneurisms in the other
present one or more of the same signs obscured by their greater depth
surface; such could only be treated of as individual cases, and will be referre
particularly in discussing the diagnosis of thoracic aneurism.

out from some central point. Thrill, either systolic or diastolic, or both, may be perceptible over the tumour; it is only very rarely however to be observed in sacculated aneurism. It may very often be found on comparison that the pulse is more feeble at one wrist than the other, or it may be obliterated on one side altogether.[1] Some hardening from degeneration of the vessels may perhaps be observed at the same time.

On *percussion*, which must always be performed with great gentleness, the note is found to be dull, over the unusual prominence; the dulness includes the sternum, extending laterally on one or both sides to gradually fade into lung resonance. It may be continuous with the cardiac dulness which is lowered or separated from it by a band of resonance, or it may encroach upon the sterno-clavicular or episternal regions. Together with the dulness there may usually be noticed increased resistance or hardness over the seat of the tumour, and this is especially marked in cases of large aneurism containing much coagulum. Some dulness may also be detected on percussion in the upper interscapular region on one or both sides.

On *applying the stethoscope* to the suspected region, the first thing which attracts attention is the impulse or systolic[2] shock, which may be intense to the ear when it is not perceptible to the eye, and only barely so to the most attentive palpation. This systolic shock may be accompanied by a bruit usually grave, rough, expansive, more distinct over the centre of the tumour than over the aortic valves. The second sound, clear and ringing at the base of the heart, may over the tumour be accompanied by a peculiarly abrupt shock or second impulse to the ear, which impulse may be even apparent also to the hand. Sometimes a diastolic bruit is audible, in which case the second shock sound is usually obscured or lost. All the auscultatory sounds of aneurism are most audible directly over the tumour; they may be conducted along the course of the aorta, and become very audible at one or both interscapular regions. Over the tumour the respiratory murmur is absent, but on passing the stethoscope aside to the acromial region, the breath-sound is found to be more or less bronchial, and the voice-sound to be more bronchial, though true bronchophony is rare. In the interscapular or supra-spinous region of the corresponding side, the respiration may also have a tubular quality. Over one lung, more rarely over both, the breath-sound has often communicated to it a peculiar sonorous vibrating quality, probably by conduction from the laryngeal stridor present.[3] The respiratory murmur is often weakened, and it may be

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[1] It is usual, but perhaps scarcely necessary, here to warn the too eager observer against mistaking abnormal distribution of radial or contraction of pupil from old iritis for signs of aneurism.

[2] In speaking of the systolic and diastolic phenomena of aneurism, I refer to those signs presented synchronously (or nearly so) with the systole and diastole of the heart respectively.

[3] Professor Stokes attaches great importance to this sign. "Diseases of the Heart and Aorta," 1854, p. 556.

completely annulled at one base, though this is rare with the ob
aneurismal tumours we have now principally in mind. Wit
laryngoscope no alteration may be found in the condition
cords, or they may be lax and act feebly with respiration, c
(usually the left) may be completely paralysed and motionles
rare cases both vocal cords have been found paralysed. The en
ment of the laryngoscope and sphygmograph would be quite
fluous in the presence of half the signs above enumerated.
become useful aids in certain obscure cases, however, to whi
shall presently refer.

Such are the signs of aneurism which may be present in ca
which the dilatation is situated near the surface, at the ascend
transverse aorta—its favourite seats. We must, before consi
the less certain signs presented by aneurism more obscurely
discuss the mechanism and diagnostic value of the more imp
of those above enumerated.

The unequal pupils, venous obstruction, local bulging of the
wall, displaced heart, percussion dulness, and the auscultatory
nomena of tubular, enfeebled, or annulled respiration, are mere pr
signs common to aneurismal or other tumours within the chest.
unequal pulses, the rhythmic pulsation, bruits, and shock sig
specially characteristic of aneurism, though some of them m
produced or simulated by solid growths.

Inequality of pupils is not a very common sign of ane
although when present it is a very striking one. The affected
is usually contracted and immovable, and corresponds with th
on which the aneurism is situated. Of thirty-six cases of aneur
the arch, of which I have notes, the pupils were unequal in fou
Dr. Walshe has observed the affected pupil vary within a few
being "now equal to, now notably, now slightly smaller, now
than the other in size," and in one of the four cases above
tioned the pupil varied from day to day in a similar manner.
cause of the affection of the pupil is admitted to be pressure
the sympathetic, paralysing it and permitting the unopposed act
the third nerve upon the pupil, or irritating it and producin
rarer phenomenon of dilatation of the pupil by excited sympa
action (Walshe). The degree of displacement of heart depends
the position and size of the aneurism. The apex beat when th
or second portion of the arch is affected is lowered and more
displaced to the left. This displacement may be extreme in ane
of the first portion, the tumour taking up the whole of the n
position of the heart, and being readily mistaken for it. The b
the heart is also lowered, so that the organ lies more transvers
the chest than natural, and there is commonly pulsation at th
gastrium, which must not be hastily received as evidence of dila
of the right ventricle. The cardiac impulse may be increased in
it is often, however, not stronger than natural, and may be enfe
It is remarkable how much less common hypertrophy of th

ventricle is than one would expect.[1] The cause of this is not clear, unless it be deficiency in the coronary circulation, for one would suppose there must be increased resistance to systemic circulation in all cases of aortic aneurism.

The heaving expansile impulse, distinct from that of the heart, is diagnostic of aneurismal tumour, which, however, must be near the surface to give this sign. It is very frequently present in aneurism of the first and second portions of the arch, and it may also be present in large aneurism of the descending arch, but only in the later stage when erosion of the ribs and vertebral processes has enabled the tumour to present as a pulsatory swelling in the left interscapular region. In cases in which the wall of the sac is greatly thickened by fibrinous laminæ, the expansile thrust may be entirely lost, and a knocking impulse alone felt which it is impossible to distinguish from that communicated to a solid tumour by the aorta underlying it. The position of the tumour and the nature of the diastolic sounds will greatly assist the interpretation of this sign. In all cases where the impulse is obscure, the plan suggested by Dr. Stokes will be found of great value, viz., to "make pressure with the flat of the hand on the anterior part of the chest, while the other hand is placed between the shoulders during expiration." By this means an obscurely and deeply expanding character of the impulse may be detected which will favour the probability of its aneurismal origin. There is sometimes to be felt a distinct thrill with the beat of the tumour. This "frémissement cataire," stated by some authors to be almost always present, is in reality not of frequent occurrence. Of eight cases in which I have myself observed marked thrill, in one accompanying also the diastole, in four the aneurisms were secondary to regurgitant disease of rheumatic origin, and they were all probably of the fusiform kind.[2]

The systolic bruit, often absent,[3] though occasionally to the experienced ear very characteristic of aneurism, is by no means, as a rule, a reliable sign, except in those cases in which it is localised at some portion of the aorta distant from the heart, as in the right or left interscapular region or along the left side of the spine. A diastolic bruit most audible at the site of the suspected aneurism, while the second sound is clear at the base of the heart, is an important sign; a murmur replacing the second sound at the base of the heart is of value in diagnosis when there is also evidence of a thoracic tumour, the nature of which is otherwise obscure; its value consisting in such a case in its indicating disease within the aorta, and so rendering the aneurismal nature of the tumour very probable. The importance of ascertaining whether a bruit replaces one of the sounds of th or is superadded to it, being, as it were, heard through it, has be pointed out by Dr. Parkes:[1] in at least a third of the cases, h of obvious aneurism of the thoracic aorta there is no brui audible, and when we take into account obscure cases the tion becomes much larger.

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[1] Hypertrophy of the heart was present in 8 only of 22 cases carefully recorded by Dr. Blakiston, in his work on diseases of the heart.

[2] Dr. Hope had never seen a case of aneurism with thrill. Dr. Walshe states it to be more common in "peripheral dilatation" than true aneurism.

[3] Lebert states it to have been present among the earlier signs in half his cases. *Loc. cit.* It existed in 22 out of my observed 36 cases, in 12 instances the bruit being double.

The peculiar diastolic shock sound when once heard, or rat by the ear, is not easily forgotten. It is, when present, most sig of aneurism, and when succeeding to a more or less distinct impulse, I believe absolutely so. It is only to be heard in a affecting the first and second portions of the aorta, and when the is very near the surface the shock is not infrequently so grea communicate a second impulse to the hand. Of the th cases I have already referred to this sign was present in fo and in ten of these there was no murmur present, although majority of them other signs of percussion, impulse, and rendered the diagnosis clear. In a few instances, howev notably in three, the diagnosis (speedily verified by death) w difficult, and depended mainly upon the importance attached ign. This phenomenon has been variously explained, and ha rise to much ingenious discussion.[2] It is no doubt of c mechanism, and is made up partly of the conducted second which is accentuated in these cases, but is chiefly cau the transmission of a wave to the surface with the closure aortic valves. The sac of the aneurism becomes fully diste little later than the aorta itself, so that the systole of the commences a trifle sooner than that of the aneurism. The valves close at the moment of aortic systole, and at the ins their closure the shock wave is transmitted through the an If there be any imperfection of the aortic valves so as to give appreciable regurgitation, the shock-sound or impulse is eit developed or very imperfectly so.

DIAGNOSIS.—Having given at some length the general sym and signs of aneurism, keeping in view more particularly thos in which the disease affects that portion of the aorta occupyi anterior mediastinum, we have to take also into account in t gnosis the question as to the part of the aorta affected by the ism, and, if possible, how to distinguish between aortic aneuris dilatation of the innominate, subclavian, carotid, and pul arteries respectively.

In aneurism of the *ascending aorta*, but beyond the heart, th are grouped about the second right space close to the sternum a centre. Greater displacement of heart, and interference w function, with cyanosis and dropsy of the upper half of the from pressure upon the innominate vein, are more often met with when the disease is in this situation. The rule is nevertheless for the aneurism to extend towards the surface rather than deeply, so that the vein often escapes serious compression. As the disease advances, the downward displacement of the base of the heart becomes more decided, and the area of pulsation enlarges downwards and to the right (Sibson). When the *transverse portion* of the arch is affected, the *manubrium sterni* is the central region of disease signs, which have a tendency to extend, however, more to the left than the right of the sternum. There is frequently no external tumour from the aneurism projecting backwards from the arch. The signs of pressure upon the air and food tubes, and their functional disturbance through involvement of the pneumogastric nerve, are most common in this variety, inequality of pulses also shows extension of the disease to this portion of the arch. In aneurism of the *descending portion* of the arch the signs usually present themselves most distinctly on the left side of the spine in the upper interscapular region, although the tumour may present at the second left space near the sternum. The pain is severe in the back and shoulder, the dyspnœa is usually pulmonary, either paroxysmal, assuming the character of asthma, or constant from pressure on the left bronchus, or partaking of both these characters. Diminished respiration with dulness at the base of the left lung is commonly to be observed. There may be partial or complete paraplegia from erosion of the vertebræ and pressure upon the cord. Aneurisms affecting the *descending thoracic aorta* are comparatively uncommon, and unless very large, difficult to detect. If very large, there may be curvature of the spine, displacement of the heart forwards and to the right, local dulness and possibly pulsation. The whole side may be dull with absence of respiration, from pressure upon the bronchus and subsequent blocking of the lung by retained secretions. A bruit localised in the back is almost diagnostic of aneurism in this situation. Persistent pain is always present, and is often the only sign of the disease. Laryngeal pressure signs are not present.

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[1] Clinical Lectures. Med. Times, Feb. 1850.

[2] See a criticism of the opinions of Drs. Bellingham and Lyons and M. Gueri Stokes's book above quoted, p. 546.

It would be a matter of great practical importance to be able to say whether an aneurism affecting the innominate, subclavian, or carotid artery involved the arch of the aorta, since, if the aorta be distinctly included, the slender hope of permanent relief which might otherwise be entertained from operative procedure is still further diminished. It may also come to be of considerable importance to know whether in a given case the disease principally affecting the aorta involves one of these main vessels at its origin, as affording the chance of temporary arrest by checking circulation through that vessel. Unfortunately, the diagnosis in both these respects is in many cases beset with the greatest difficulties. There is no absolutely diagnostic sign separating aneurism of the innominate (the vessel with regard to which the question most often arises) from that of the aorta. If, however, the aneurism has its centre of pulsation or other signs below

the second rib, or if it encroach upon the sternum without al
senting behind the sterno-clavicular articulation and the epi
notch, the presumption is, that the arch of the aorta is de
involved. A well-marked shock-sound would be also very sig
of the disease being mainly aortic. We may also get some in
tion from a comparison of the state of the pulse at the two
and this naturally leads us to consider the value of the sphygm
in the diagnosis of aortic aneurism. For the purpose of show
kind of information yielded by the use of the sphygmograph,
take one or two tolerably well-marked cases.

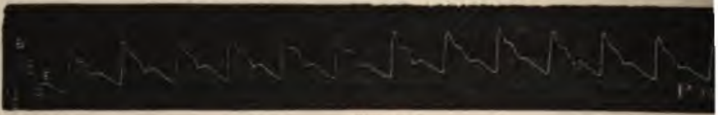

FIG. 1.

In Case I., that of Eliza B——, from which the above
tracing (Fig. 1) was kindly taken for me by Dr. Burdon San
there was a circumscribed aneurism of the ascending aorta, p
ing its distinguishing signs—local bulging, dulness, systolic im
and faint diastolic bruit—at the second right intercostal spac
the sternum. Here it will be seen that the systolic wave, A
prolonged, occupying a greater proportion of the tracing t
should do—*i.e.*, there is a prolonged effort on the part of th
ventricle to overcome increased resistance. The impaired elasti
the arteries is also shown by the blunted, elbow-like point
at the commencement of the systolic upstroke. The sho
percussion wave, A B C, of the pulse, is at the same time
what diminished; but as this phenomenon is general, not lim
one radial, it has no significance, since it may be produced by
other conditions; it is, however, very commonly, but by no
constantly, met with in aneurism of the main vessel. The
evidence we gain in this case, therefore, is that of the prese
arterial disease.

Case II., in which the tracings were also taken by Dr. Sand
Dr. Murchison has kindly allowed me to make use of to illu
some further points respecting the use of the sphygmograph. (F
In this case the pulsations were different in the two radials.

Thomas J——, a coachman, was admitted into the Middlesex
pital, under Dr. Murchison, in November 1868, with a puls
tumour in the chest, most prominent at the third right inter
where a double bellows murmur was audible. There was scarcel
difference to be detected by the finger in the two pulses, but the
was thought to be slightly the smaller. It was for this reason
there being no doubt about the diagnosis, that Dr. Sanderson
requested to take tracings of the two pulses.

In the left radial tracing, the lengthened systole, A C D, is the noticeable feature; in the right, on the other hand, we have superadded to this sign of general increased resistance to circulation, others indicative of local interference with the arterial movements. The systolic vibration, or percussion wave, A B C, very marked (rather exaggerated) in the left pulse-tracing, is almost lost in the right.

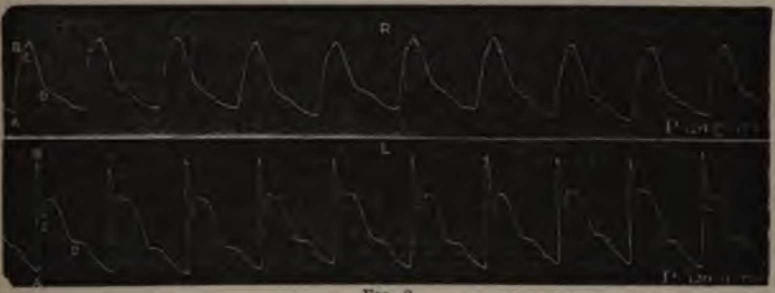

FIG. 2.

The dichrotism is also more distinct in the left than the right. At the same time the right pulse is not notably smaller than the left. Hence from the pulse alone we find evidence (1), of arterial disease, in the prolongationof the systole; (2), of a tumour pressing upon or dilatation of the artery, in the local effacement of vibratile impulse (percussion wave) in the right radial, without diminution in the size of the pulse.

Had this latter phenomenon been due to extension of a coagulum, so as to partially occlude the innominate, the pulse would have been much smaller on the right side. But a saccular dilatation of the vessel, or a solid mass in contact with it, might act as an inefficient damper in destroying the vibratile shock, without affecting the size of the pulse.

In this case the aneurismal tumour invaded the thoracic parietes, and presented to the right of the sternum, but the patient died of an intercurrent attack of pneumonia. After death, the aneurism was found to be globular, as large as the two fists, and involving the whole circumference of the aorta, from its commencement to the origin of the right subclavian, which came off as a separate trunk. The aortic orifice was incompetent.

It has been before observed that evidence of arterial disease coexisting with that of a tumour within the chest strongly favours the presumption that the tumour is aneurismal. Thus the intelligent use of the sphygmograph may prove in certain cases of considerable value in helping the diagnosis of aneurism, although it should be clearly understood that alone it is nearly useless for that purpose.[1]

Another sign of aneurism, much insisted upon by some authors, is *postponement* or delay of pulse in one wrist. Strictly speaking, it is not correct to say that the pulse is postponed, for it begins a at the same time in the two wrists; but the sense of delay conveyed by the finger through the initial shock or percussio being obliterated on one side, while it is well marked on th Thus, in Case II., we can readily understand that the shoc A B C, constituted the pulse wave, as appreciated by the finge observer when applied to the left radial, while on the right s wave was annulled, and the true systolic wave, A C D, alone ciable. Hence the pulse on the right side might have app the observer postponed (this point is, however, not entered notes of the case). This point of delay in the pulse, then, i present, of some practical value in diagnosis; it means the a tion by the finger of the deficiency of shock wave on one side

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[1] In the Med. Times and Gazette for 1873, pp. 141 and 122, will be found valuable original papers, by Dr. F. A. Mahomed, on the use of the sphygmograph in the diagnosis of aneurism.

The distinction of aneurism of the thoracic aorta fro tumours in the same situation, from mediastinal abscess, lo pyema, uncovered aorta from contracted lung disease, dilate pericardial effusion, aortic valve disease, laryngitis, asthn angina pectoris, requires a few further remarks.

The leading features which separate aneurism from other tinal tumours have already been incidentally referred to in s of the value of the individual signs present in aneurism, and ha more specially considered in the diagnosis of mediastinal t We may, however, briefly mention a few other considerations importance to bear in mind in cases of difficulty, as affordin taken in association with the signs of tumour, additional evid or against aneurism.

1. If the age of the patient be under twenty-five, in the of any history of direct injury, the chances are against aneuri

2. Great emaciation, in the absence of intense prostratin is against aneurism.

3. Great displacement of heart, in the absence of marked a large pulsating tumour, is against aneurism.

4. Female sex of the patient is against aneurism.

5. On the other hand, severe pain, constant, with occasional bation, is in favour of aneurism.

6. The more inconstant the distal signs of pressure—unequa irregular pupils, laryngeal and bronchial dyspnœa, dysphag greater the probability of the disease being aneurismal.

7. Dr. Walshe observes that "the absence of symptoms an indicative of ordinary affections of the heart and lungs, in dividual suffering from persistent anomalous disturbances wit chest, even though he does not, or rather because he does not, any failure of general health, affords strong motive for sus aneurism." In the diagnosis of pulsating empyema from an very rarely required, the presence of fluid impulse without b shock sound, the extension probably of the effusion beyond th of an aneurism, the pointing of the abscess, and the presence o tive fever, would be the distinguishing signs.

Cases not infrequently occur of disease affecting the apex of one lung, causing the retraction of its margin away from the base of the heart and aorta. Such cases present many of the signs of aneurism—dulness, forcible local pulsation, sometimes a bruit, with palpitation and dyspnœa.[1] The history of such a case, the evidence of distinct disease of the lung, and of enlargement of the opposite lung, with elevation rather than depression of the heart's apex, and the absence of other pressure signs, will usually prevent error in diagnosis.

In some cases, aneurism of the descending aorta, compressing the left bronchus, and leading to collapse of the lung, or to its blocking by catarrhal products, becomes so masked by the secondary affection, and from the depth of the tumour in the mediastinum, as to make the diagnosis from chronic pneumonia or pleurisy most difficult. In such cases, however, the urgency of the dyspnœa, especially on excitement, and the deep-seated pain and palpitation, will usually awaken at least grave suspicion in the right direction.

Dr. Hope lays some emphasis on the possibility of aortic regurgitant disease being mistaken for aneurism. The symmetrical pulsation of all the vessels and the seat of the murmur will usually prevent such an error. Dilatation of the commencement of the aorta is not unfrequently occasioned, however, by regurgitant disease, and the locomotion of the vessel being always increased, the signs of the dilatation are thereby much exaggerated.

Laryngeal symptoms are apt to be present in the most obscure forms of aneurism, projecting deeply from the back of the arch. Such cases may be mistaken for laryngitis; but the inconstancy and paroxysmal character of the symptoms, so rare in adult laryngeal affections, should at once arouse suspicions, and laryngoscopic examination with a more minute exploration of the chest will then usually solve the difficulty.

PROGNOSIS.—In aneurism of the aorta, the prognosis is within a brief and uncertain space of time fatal. It is the uncertainty as to the time of death, and the suddenness with which it may at any time occur, that gives to this disease its peculiar terror. It is this peculiarity, moreover, which should make us so extremely careful not to make a wrong diagnosis; and when there is any doubt, as there often must be, we should give the patient or his friends only a sufficient insight as to his critical condition to insure the due settlement of his affairs.

Duration.—From six months to four years from the time of detection of the aneurism (Lebert).[2] This limit has been extended in certain cases. The main characteristic of the duration of aneurism, however, is its uncertainty. The aneurismal wall may be obviously thickening by fibrinous deposit at one point while fatal erosion is taking place at another.

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[1] I have met with several cases of the kind which might readily have been mistaken for aneurism; and in one case the error had been actually made, and the diagnosis of aneurism communicated to the patient by an observer of some experience.

[2] Loc. cit. Analysis of 30 cases.

Mode of Death.—Rupture is by far the most common ter
of aneurism, and the more latent the aneurism, the more u
does death occur in this way. Dr. Sibson[1] remarks, "It
observed throughout, that the greater the proportion of rupt
any group of aneurisms of the aorta, the smaller is the pr
of instances in which the disease is indicated by symptoms
life, and *vice versâ.*" The rupture may take place into the peric
left or right pleura, bronchi or lungs, trachea or œsophagus
ternally or more rarely into the vena cava, right auricle, &c
secting aneurism, most commonly affecting the first portion
aorta, almost invariably bursts into the pericardium. Circu
aneurism of the ascending aorta most commonly also gives
the pericardium, of the tranverse aorta into the trachea or œso
of the descending into the left pleura, left bronchus, or œsoph
into the abdomen. A certain small number of patients die
haustion, fewer still of syncope or asphyxia (apart from hæmo
or from acute intercurrent disease.[2]

TREATMENT.—The objects we have in view in the treat
thoracic aneurism are, firstly, to diminish the strain upon the
vessel as much as possible; secondly, to encourage the de
of fibrine within the aneurism. We have further, by the a
tration of anodyne remedies internally or locally, to lessen
ing. In carrying out these objects, rest as far as is possible
circulation is of the first importance, and the conditions most
sary to secure this rest to the circulation, are strict muscular
mental quietude, and regulated diet. It requires much intel
on the part of the patient, and tact on that of the physician, t
tain these conditions for a sufficient length of time to be of
even when surrounding circumstances are favourable.

The recumbent posture, or that most nearly approaching it
comfortable to the patient, should be preserved, all excitement a
and the nutrition maintained by a diet restricted in quanti
enriched in quality. Blood rich in nutritive elements more
deposits fibrine, and favours those efforts at repair which resu
welding together of the fibrinous layers nearest the arterial
while, mechanically speaking, it has no more pressure effect u
weakened vessel than blood deficient in these elements. On the co
we daily find that whenever an anæmic condition is present the
lation is hurried and more easily disturbed. Evidence of a rapid
ing down of the laminæ which had almost blocked an aneuris

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[1] The Aorta and the Aneurisms of the Aorta.

[2] See on this point the statistics of Dr. Crisp, Dr. Sibson, and Professor Lebe
above general remarks are more especially founded upon the very careful and
tables of Dr. Sibson—584 cases. Lebert's statistics are on this head less trustwo
numbers being much smaller; thus of 41 cases he finds 4 rupture external
Crisp and Sibson finding this termination in about 4 per cent. Lebert also
cases of rupture into œsophagus, whereas this is rather a frequent termination
of aneurism—of transverse and descending aorta—about 5 per cent. (Sibson).

sometimes be found *post mortem* in those who have subsequently to careful treatment again been subjected to the debilitating circumstances of a life of poverty. Fluids must be only very moderately partaken of; milk, soft puddings, eggs, and a moderate quantity of meat may be allowed. Mr. Tufnell,[1] whose treatment of large internal aneurisms has been remarkably successful, restricts the food taken to 2 oz. of bread-and-butter and 2 oz. of new milk for breakfast; 2 or 3 oz. of bread and 2 or 3 oz. of meat for dinner with 2 oz. to 4 oz. of milk or claret; and 2 oz. of bread-and-butter with 2 oz. of milk for supper. In his first recorded case he maintained this treatment, combined with absolute rest, for nearly two months with complete success. Mr. Tufnell would of course allow a certain slight variation from this diet measure to suit individual cases: his treatment is moreover only applicable to cases of sacculated aneurism. Dr. Sibson[2] also advocates the regulation of the diet in conjunction with rest, but is content to limit the quantity of fluid taken *per diem* to within one pint. Let me repeat, rest in the recumbent posture is of the utmost importance to the success of this treatment; Mr. Tufnell reckons that in some of his cases this alone lessened the number of distensions of the aneurismal sac by more than 20,000 a day![3] Stimulants, or I should rather say stimulation, must be absolutely interdicted. During this treatment too frequent examinations are to be avoided, but the circulation and the aneurismal impulse, when within our reach, should be carefully watched. Certain drugs—chloral, opium, digitalis, veratria, and aconite—are useful to allay excitement of circulation. They are none of them efficacious without rest, but they, and particularly chloral and opium, may help in diminishing the restlessness and impatience of persons naturally irritable who have great difficulty in submitting to the treatment by diet and recumbency. Belladonna applications may be employed for the purpose of relieving pain, but when this is acute the subcutaneous injection of morphia is the best remedy. The continuous application of an ice bag, suspended from above the patient, to the tumour when prominent externally will often greatly relieve pain, reduce inflammation, and perhaps even help to promote consolidation within the sac. Gentle laxatives or saline purgatives must be given to prevent any effort in relieving the bowels. When, as sometimes happens on the patient first coming under notice, there is any undue fulness of vessels present, free purgation with salines will be attended with marked relief up to a certain point. In cases of urgent dyspnœa with engorgement of vessels, venesection should be promptly employed; repeated blood-letting after the manner recommended by

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[1] Thoracic Aneurism successfully treated by restricted Diet and the application of Ice. Dublin Hospital Gazette, January 1858; also on the Treatment of Thoracic and Abdominal Aneurisms, by J. Tufnell, F.R.C.S.I., Army Med. Rep. for 1862, and Medico-Chir. Transactions for 1874.

[2] Croonian Lectures, Lancet, 1870.

[3] See an able review of the modern treatment of aneurism in the Medical Times and Gazette for December 20, 1873.

Valsalva[1] in the last century, combined with the lowest diet, is not likely to find favour in the present day.

Various internal remedies have been administered with tl of favouring the formation of coagulum within the aneurisn by rendering the blood less watery—*e.g.*, saline purgatives, di or by affecting it or the aneurismal wall in some specific wa acetate of lead, iodide of potassium, ergot. The free admini of iodide of potassium in aneurism has been pretty exte tried. It seems to have been first introduced by Nelat 1859, and subsequently tried by Drs. Bouillaud,[3] Chucke of Calcutta, Roberts[5] of Manchester, and Balfour[6] of Edi and the concurrent testimony of these several observers favourable to the drug as a valuable agent in the treatment disease. Dr. Balfour holds that "no treatment for aneuri especially for internal aneurism, holds out anything like ar prospect of relief, if not of cure, with that by the iodide of pota The drug must be given in large doses (gr. xx) and contin many months. Its mode of action is unknown; Dr. Balfour[7] that it is not by increasing the coagulability of the blood, bu sedative action upon the heart and "by some peculiar action fibrous tissue, by which the contraction of the sac is aided walls are strengthened and condensed."

Langenbeck[8] having observed the great value of ergot, part when used subcutaneously as ergotin in arresting hæmorrhag the drug in the same way in aneurism, and with considerable in a case of the disease affecting the innominate and sub trunks. He injected ½ gr. of the watery extract, increasing t every three days. It is difficult to say how far in this, as i instances of the reputed efficacy of drugs in the treatment of latory diseases, the attendant rest may have aided in produc amelioration of symptoms.

Rest and recumbency, with the aid of drugs, failing or impracticable for the obliteration of aortic aneurism, the tion arises, whether any operative measure can be a to control the circulation through the sac so as to fav gradual obliteration by laminated clot. M. Velpeau,[9] in seems to have been the first to entertain the question of liga one or more of the main branches of the aorta for the cure of aneurism, and this treatment has been most recently advocated by Dr. Cockle[1] and practised by Mr. Christopher Heath.[2] The exact value of this method of treatment, and the rules which should guide us in adopting it, cannot be regarded as yet thoroughly ascertained. In cases in which one of the great vessels—most commonly the innominate—proceeding from the arch is largely involved, or in which the aneurism is at least extending in the direction of one of these main branches while there is no evidence of extensive arterial degeneration, the distal ligature may be most appropriately tried, the principle of the ligature being to greatly enfeeble the current through the sac by arresting the circulation through the branch principally involved or most nearly proceeding from the aneurism. Ligature of the right carotid or subclavian, or both, or of the left carotid, must be decided upon according to the direction of the growth of the aneurism. Distal pressure upon the great vessels of the neck has been employed, and though most difficult to effectually carry out, is yet, perhaps, worthy of a further trial.

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[1] Vide Observations on Aneurism, by Mr. Erichsen, Syd. Soc. 1844, pp. 239

[2] Clinique Européenne, July 1859.

[3] Idem, August 1859.

[4] Iodide of Potassium in the Treatment of Aneurism. Brit. Med. Journal, Ju

[5] Clinical Lecture on the successful use of Iodide of Potassium in the Treat Aneurism. Idem, January 1863.

[6] On the Treatment of Aneurism by Iodide of Potassium. Edinb. Med. July 1868.

[7] Idem, January 1874, p. 6

[8] Idem, November 1869. Abstract from the Berliner klinische Wochen March 1869.

[9] For the most modern authoritative information on the surgical treatn aneurism, *vide* Lectures delivered by Mr. Holmes, at the College of Surgeon Lancet, July 1872, lect. ii.

The common object of the different kinds of treatment we have hitherto considered, viz., rest, diet, compression, and ligature, has been that of encouraging spontaneous coagulation within the aneurismal sac, whether by lowering the circulation generally, or by locally arresting it entirely, or lessening its force or altering its direction.

Mr. Moore[3] in 1864 proposed a new treatment of aneurism, by the insertion into the sac of a foreign body (fine iron wire) to act as a nucleus for rapid and firm coagulation. The result in the case to which he applied this treatment—a case of aneurism of the thoracic aorta rapidly perforating the chest-wall—was discouraging, inflammation of the sac, embolism of distant vessels, and great increase in the sufferings of the patient being occasioned. The same idea has since, however, found favour among a few bold operators, who have severally tried needle punctures, the introduction of carbolized catgut, watch-spring (14 inches!). and iron wire, with uniformly deplorable results. The latest and most rational method of the kind adopted is that of Dr. Levis of Philadelphia,[4] who has introduced through a capillary trochar a great length of fine horsehair into the sac of a subclavian aneurism. I think it must be allowed, however, that this mode of treatment is wrong in principle, and must, therefore, in the majority of instances fail in practice. If direct coagulation of blood within the sac be aimed at, it is only thus accomplished in a dangerous and imperfect

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[1] Further Contributions to the Pathology and Treatment of Aneurismal Tumours of the Neck and Chest, by John Cockle, M.D. Lancet, 1869, pp. 422 and 489. See also Clinical Transactions for 1872.

[2] On the Treatment of Intra-thoracic Aneurism by the Distal Ligature, by Christopher Heath, F.R.C.S., 1871.

[3] On a New Method of procuring the Consolidation of Fibrine in certain Incurable Aneurisms. Med.-Chir. Trans., vol. lvii., p. 129, *et seq.*

[4] Referred to by Mr. Holmes, in London Medical Record, December 17, 1873.

manner, the clot formed being subdivided and soft, and entangle
a foreign body in the centre of the sac instead of being lamina
firm, and deposited around its circumference; hence dange
flammation of the sac, of capillary or larger embolisms, and o
poisoning from rapid disintegration of the clot. If the obje
determine coagulation within the sac by setting up a certain d
inflammatory action, or if the fear of inflammation resulti
these procedures be disregarded on this ground, the practi
equally be condemned as dangerous, if not unjustifiable, for
no means of controlling the inflammation when induced in th
blood-channel. It must further be borne in mind that by pr
suddenly coagulation within an aneurism without that local or
lowering of the circulation which would favour its natural occ
we necessarily run great risk of causing rapid extension of the
in some other, perhaps more fatal, direction. These objections,
be confessed have, too, a certain force when applied to the so
less modern practice of galvano-puncture, which has rece
common with all other methods of treating this deadly malad
very keenly discussed and tested. There is this striking ad
in galvano-puncture, however, that no foreign body is allo
remain within the aneurism. It has, moreover, been trie
success now in a few instances.[1]

The idea of employing electricity in the treatment of ar
seems to have sprung from the physiological experiments of
more 1824, Müller 1832, and Ansell 1839, and to have be
applied in practice by M. Pétrequin in 1841.[2] It was Ci
however, who, in 1846, first employed electro-puncture in the
ment of aortic aneurism,[3] and the first definite principles on w
proceed in the employment of this agent were laid down still l
MM. Baumgarten and Wertheimer in 1852,[4] as the result of ex
experimental inquiry. The experiments of these gentlemen sho
that coagulation might be with certainty induced at any point
a large vessel of an animal during life by electrolysis, (*b*) th
coagulation was most firmly [5] affected around the positive needl
both were introduced; (*c*) that the best way, however, of pro
coagulation was to insert the positive needle only, applyi

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[1] Of 13 cases treated by Ciniselli's method, between July 1868 and July
were "cured." Of them 3 relapsed after 17, 3, and 4 months respectively; t
case, however, after a second operation, being again "cured" and remaining s
months. For details of 23 cases, and for a description of Ciniselli's method of en
electro-puncture, see his paper in the Annali Universali di Medicina for Novem
p. 292, *et seq.*, Table, p. 625. An abstract of this paper is given in the Jahresbe
1870, Bd. ii. s. 109.

[2] Vide Essay by Dr. Fraser, of Edinb., on the Action of Galvanism on Blood
Albuminous Fluids, 1867.

[3] Case related in the Annali Universali di Medicina, 1870, p. 294, and referred
22 others in a Table at p. 625.

[4] Gazette des Hôpitaux, June 19, 1852.

[5] Confirming a previous observation by Prof. Schuh, Vierteljahrschrift für d
tische Heilkunde, Bd. i., 1851.

negative element by means of a moistened sponge or metallic conductor placed on the external surface.

Dr. Fraser, in 1867, confirmed and added to these conclusions, observing that, whereas at the positive needle a firm and comparatively small clot was formed, at the negative needle, on the other hand, the coagulum was large and frothy. Dr. John Duncan[1] is, however, of opinion that the needles connected with both poles should be introduced, and in this view he agrees with Ciniselli.[2] Dr. Althaus,[3] on the other hand, regards the negative pole as the one to the use of which "we have to look for the cure of aneurism."

As to the mode in which electricity effects coagulation within an aneurismal sac, the question whether it may be by the mere passage of the electrical current through the blood, is set at rest by the observation of Dr. Fraser, that although the two needles be separated by an interval of blood through which the current must pass, yet coagulation takes place only around each needle separately. A certain amount of inflammation is often set up in and about the sac by this agent, which no doubt, if it do not proceed to a dangerous extent, may help to promote consolidation; this inflammation is, however, shown by Drs. Duncan and Fraser to be not essential to coagulation, but, on the contrary, to be guarded against as one of the chief sources of danger. The coagulation is produced in truth, as pointed out by Steinlein[4] and confirmed by others, by (*a*) electrolytic decomposition of the salts of the blood which are mainly instrumental in maintaining its fluid state; (*b*) by the acid elements set free at the positive pole directly causing coagulation there, or combining with the oxidised metal to form salts which precipitate the albumen. This latter action may be increased by coating the positive needle with some more oxidisable metal, such as zinc.

In the writings of Dr. John Duncan, Dr. Fraser, Mr. Holmes,[5] and Prof. Ciniselli already quoted, will be found recorded the clinical and physiological experiences which furnish us with all the knowledge we at present possess on the subject. The valuable records of individual cases of galvano-puncture in aneurism which are gradually collecting are as yet too few to admit of classification. So far as they have

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[1] On the Surgical Applications of Electricity, Edinb. Med. Journal, 1872, 506, *et seq.*

[2] In this view also Mr. Marcus Beck concurs; and the comparative effect upon an albuminous fluid of inserting only one or both needles is well illustrated by a simple experiment made by Mr. Beck and Dr. Poore. Into some white of egg held in the hollow of the hand the positive needle connected with twenty cells of Weiss' battery was introduced, a sponge connected with the negative pole being applied to the back of the hand. After five minutes no action upon the albumen had been effected. Both needles were then introduced together into the albumen, and in two minutes a firm coagulum of the size of an oat surrounded the positive needle-point, and a frothy mass as large as a pea was found at the negative needle. Lancet, 1873, p. 550.

[3] Medical Electricity, 2nd edit., 1870, p. 607.

[4] Galvanopunctur bei Varicositäten und Aneurysmen—Zeitschrift der k.k. Gesellschaft der Aerzte zu Wien, 1853. Quoted at p. 471 of Dr. Hammond's translation of "Meyer's Electricity in its Relations to Practical Medicine."

[5] Lancet, 1872, vol. ii., pp. 336 and 663.

gone, however, they do not encourage us to anticipate a very f
opinion of the operation, for of nine cases of aortic aneu
treated of which accounts have appeared in the English
within the last two years, in only one, that of Dr. McCall A
has a decidedly good result been obtained, although in one or
instances, in Drs. Ralfe and Johnson's case, and in a case of Dr.
perforation of the chest wall has been for a time averted.
instances, however, this perforation has undoubtedly been ha
the needle punctures. Appropriate cases in which the ope
been undertaken with the view of curing the aneurism must
fied apart from those in which the puncture is made with
of temporarily stanching an aneurism already diffused and
ing to burst through the surface, before we can hope
reliable statistics upon which to decide as to the true valu
treatment: moreover we certainly have not yet arrive
method of operating agreed on all hands as the best. Mean
following appear to be the most important points respe
operation which the physician should bear in mind:—

1. (*a*) The cases of thoracic aneurism which seem most su
treatment by galvano-puncture *with a view to permanent*
those in which the dilatation is presumably sacculated, ne
surface, and advancing outwards, the sac being as yet entire
operation may sometimes be performed as a palliative m
retard rupture through the surface, or to relieve suffering.

2. The treatment by rest, with the aid of restricted diet a
priate drugs, must have first been fairly tried and proved
impracticable before any operative procedure is justifiable
same absolute rest and careful diet must be maintained th
the operation and the subsequent treatment.

3. It is best not to freeze the surface before puncture, alt
sensibility may be deadened by cold. Sometimes the operat
be usefully preceded by the administration of a dose of
subcutaneously to enable the patient to remain without suf
one position during its performance.

4. The battery used should be of many (10 to 30) cells wi
of small surface. By diminishing the size of the plates and
ing the number of the cells used, a given electrolytic power is
with a less intensity of heat than with fewer cells and larger

5. The needles connected with both the positive and negati

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[1] Foveaux's improvement of Weiss's instrument is now considered the best.
has, however, electrolysed white of egg by employing a Pulvermacher's chain o
elements moistened with dilute sulphuric acid (one part to thirty); and he su
producing in an hour a firm clot at the positive electrode three-quarters of an
of the diameter of a goose-quill and weighing nine grains. Dr. Poore sugge
worth a trial, the employment of a similar battery in the treatment of aneu
thinks it possible that patients might be able to bear the application of a cu
such a battery for far longer periods than when elements of larger surface are
of. This is of course a question only to be decided by direct experiment.
book now published.

and of the dimensions of medium-sized hare-lip pins, insulated with vulcanite to within a certain distance of their points (according to the size of the aneurism), should be thrust vertically into the aneurism so as to avoid scratching or puncturing its inner surface at any other point.

6. The electrolysis should be continued for twenty minutes or half an hour, or until some decided alteration in the pulsation or bruit of the aneurism has been produced. The withdrawal of the needle must be effected with the utmost caution, to avoid the loosening of the clot should it happily have become adherent to the wall of the aneurism. A second and a third employment of the agent may be made at intervals of several days.

The question of *tracheotomy* occasionally comes before us in cases of thoracic aneurism, in which death is threatened from paroxysmal dyspnœa. If we accept the views lately advocated by Dr. Bristowe, that not only the constant, but even the paroxysmal dyspnœa is in the majority of cases due to direct pressure upon the trachea, we can expect no relief from this operation. If, however, the view that the paroxysms of dyspnœa may be occasioned either by irritation or destruction of the recurrent nerves be still tenable, we may expect to prolong life and to render death less terrible by its performance. There have been cases in which the operation has distinctly though only temporarily saved life.

SPONTANEOUS RUPTURE OF THE AORTA.

Spontaneous rupture of the aorta is most commonly the result of antecedent disease of the coats of the vessel, more particularly of the senile atheromatous kind. It may occur, however, in consequence of stenosis of the vessel (Rokitansky), though such cases are necessarily very rare. Even in such cases, too, the proximate cause of the rupture is disease of the vessel wall, either abnormal delicacy and thinness (Rokitansky) or atheroma the result of heightened blood pressure. Rupture of the aorta most commonly takes place at or near its commencement (Broca, Peacock). It may at once extend through all the coats of the vessel, causing immediate death from hæmorrhage, or, as is more commonly the case, it extends through the internal to the middle coat, and the effused blood tearing apart the layers of this coat, or separating it from the external for a greater or less distance, a *dissecting aneurism* arises. The coats of the vessel may be thus dissected or separated apart throughout the whole length of the aorta, and even along some of its main branches. Dr. Todd[1] relates a case where the separation extended from half an inch beyond the valves to the abdominal aorta and along the innominate and right carotid arteries, causing

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[1] Med.-Chir. Trans., vol. xxvii., 1844.

drowsiness and partial left hemiplegia by compressing the cana
the last-named vessel.

There is frequently no discoverable exciting cause of the rupt
in a few cases it has been traceable to great mental emotion, o
cardiac excitement from over-distension of the stomach; exte
violence, as the shock of a blow or fall, may also give rise t
Rupture of the aorta may occur at any age after thirty. Dissec
aneurism occurs mostly in advanced age; it is about equally c
mon in both sexes.[1] There are no special symptoms attributab
rupture of the aorta save those of fatal syncope. In cases, how
in which the rupture is incomplete and a dissecting aneurism oc
the symptoms are, acute rending pain in the præcordial region
tending to the left shoulder and spine, severe cardiac dysp
orthopnœa, with profound shock to the system, pallor of countena
great anxiety, and feeble, irregular pulse. The rupture of the exte
coat may quickly follow that of the inner, and death immediately e
or the patient may rally for a few hours, or even days, before a se
attack proves fatal. In some rare cases, in which the aneu
becomes circumscribed, its future course may closely resemble th
other circumscribed aneurisms. The treatment of aortic ruptur
dissecting aneurism is, of course, merely palliative.

NARROWING OF THE AORTA.

In certain rare instances there is a congenital deficiency in
calibre of the whole aortic system. This general narrowing us
affects mainly the descending aorta, particularly the descending por
of the arch. It is most common in females, and is often overlo
being attended with no marked symptoms until the period of pub
when the insufficient general development, with marked
ciency in the sexual system, become apparent. The only phy
signs present are those of hypertrophy and dilatation of the left
tricle, and smallness of pulsation in the abdominal and iliac art

A more common form of congenital narrowing of the aorta, is
in which the constriction is limited to that portion of the arch be
the subclavian artery, either at or a little above or below the *d*
Botalli. The term "coarctation of the arch of the aorta" is
applied to this condition. The constriction rarely occupies
than half an inch of the length of the vessel. It may be ring-
as though the vessel had been tied by a moderately thick pie
string, or it may be caused by a fold, more or less deeply projecting
the vessel, or by a scar-like contraction corresponding with the
tion of the duct; or again, the vessel at this point may be conv
into a thickened, impermeable cord. The degree of constri
varies from complete closure to a diameter of three or four lines,

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[1] Rokitansky's cases show a slight preponderance on the male side; in Dr. Pea
it was slightly more frequent among females.

mere narrowing. Only in five cases out of forty collected by Dr. Peacock was the obliteration complete. The walls of the aorta at the seat of the contraction may be natural or thickened or thinned. The *ductus arteriosus* is usually closed, but in some cases it is open for part of its extent, in others it is more or less pervious throughout; it is affected in one of these ways in about one-sixth of the cases. Above the constriction the aorta is as a rule widened, sometimes greatly dilated; it may, however, be of natural dimensions; below the constriction, it is either of normal size, or more frequently somewhat diminished: occasionally, however, it is even widened, and in one case quoted by Dr. Peacock, an aneurism was found immediately below the stricture. A deficiency in the number of aortic valves has been found in one-eighth of the cases, and other cardiac malformations have been mentioned. The great branches of the arch are always enlarged, and by the communication of some of the branches derived from them (the transversalis colli, superior intercostal, internal mammary), with corresponding branches from the descending aorta (intercostal, epigastric), a tolerably free collateral circulation is maintained. Hypertrophy of the left ventricle is a necessary consequence, and is often attended with dilatation and with, no doubt, secondary dilatation and hypertrophy of the right cavities of the heart.

This affection is three times more frequent in the male than the female sex. In almost every case the deformity is either congenital, or acquired in the first few days of infant life: death however may occur at any age, usually but not always preceded by symptoms of heart disease. There are several theories to account for the occurrence of this deformity of the aorta, of which three are admitted by different authors as applicable to certain cases:—

1. Although the normal process of closure of the *ductus arteriosus*, which occurs within the first week or ten days of life, is simply one of gradual withering and contraction, yet in some instances it is delayed by the formation within the duct of a fibrinous coagulum, which may extend into the aorta and completely occlude it at the point corresponding with the entry of the duct. As the clot subsequently becomes gradually absorbed, the walls of the aorta and those of the duct contract upon it to their complete obliteration. Foerster adopts this theory as applicable to those cases in which both the duct and the aorta are completely closed.

2. In other cases, however, the coagulum may not extend beyond the duct, the walls of which, thickened from the irritation attendant upon the presence of the clot, contract upon it as it becomes absorbed, puckering the adjacent walls of the aorta in an irregular and scar-like manner, so as partially to constrict the vessel.

3. A more generally applicable explanation is, that this deformity is a partial preservation of the fœtal condition by which the aorta conveys blood to the head and upper extremities, and the pulmonary artery, continuous through the duct with the descending aorta, supplies the lower extremities and abdominal viscera. It is the view of

Reynaud and Rokitansky, and is adopted by Dr. Peacock and m
modern authors, and may be described as follows :—

At the termination of fœtal life, with the expansion of the lu
the blood-stream is diverted from the *ductus arteriosus* through t
two branches of the pulmonary artery. The blood thus diver
returns to the left ventricle and increases the volume to be tra
mitted through the first and second portions of the aorta by t
amount destined for the supply of the lower half of the body.
the normal condition of things the isthmus of the aorta,—that p
tion connecting the great brachio-cephalic trunk with the *duc*
arteriosus as it joins the descending aorta—now rapidly expands so
to become a part of the main arterial channel, while the starved duc
dwindles. If, however, as sometimes happens, whether from
fective nutrition, rigidity from inflammatory thickening, or
other cause, this *isthmus* fails to widen, the increased volume of bl
finds exit through collateral channels into the descending aorta,
branches of the arch expand, and their twigs, communicating w
branches from the descending trunk, enlarge so as to supply to
the blood required for the lower limbs and viscera. Thus far the p
cess takes place in the first days of extra-uterine life, but
narrowed portion of the aorta, having the extra pressure of blood t
removed from it, still further atrophies from disuse, and in cours
time may become quite closed, although its channel is probably
these cases never completely obliterated. An increased musc
power of heart is required to compensate for the increased resista
to circulation necessitated by the transmission of the blood thro
circuitous and divided routes instead of by one short and br
channel to the descending trunk.

It has already been stated, that this defect in the aorta is
necessarily attended with any symptoms, and that when they a
they are those of lesion secondary to the aortic deformity, of hy
trophy and dilatation of the heart, rupture of the heart or aorta
great dilatation of the main vessel above its narrowed portion.
is unnecessary to refer to these symptoms in detail.

DIAGNOSIS.—The diagnosis has not often been made, but when at
tion is directed to such a case the physical signs pointing to the
striction and its situation, though few, are tolerably significant. T
are those of hypertrophy of the heart, most distinctly of the
ventricle, usually attended with some dilatation, throbbing of
great vessels of the neck and upper extremities; while arteries
usually visible, the transverse cervical, and thyroid, and the s
anastomosing vessels at the margin of the sternum and epigastr
are enlarged, tortuous, and pulsating. With this activity of circula
in the upper half of the body is contrasted the feeble pulsatio
the abdominal aorta, iliac and femoral arteries, all pulsation in
popliteal and the tibial vessels being often absent. A systoli
rather post-systolic, murmur may be heard over the aorta and

ver the enlarged vessels. The presence of aneurism or of some ther mediastinal tumour pressing upon the aorta will be excluded by the absence of any other signs of pressure upon the nerves, or food or air tubes, or upon the lungs.

PROGNOSIS.—In a considerable proportion of cases death occurs from some lesion apparently altogether unconnected with the aortic deformity, eleven cases out of the forty referred to by Dr. Peacock having died from cerebral disease, bronchitis, pneumonia, &c. But the circulation is carried on at high pressure, at least as regards the heart and first portions of the aorta, and sudden death, from syncope or rupture of the heart or aorta, occurred in eight of the forty cases. In the rest, the heart in time gives way under its excessive toil, degeneration and dilatation succeed to hypertrophy, and death slowly occurs in the way usual to such affections. Supposing, therefore, that the diagnosis be made while the patient as yet appears to be in fair health, there is a very fair chance that if, bearing in mind the very mechanical nature of the result of this malformation, he be warned against such exercises as increase the circulation, and encouraged in calm intellectual pursuits and a sedentary profession or business, he may possibly live to the average period of life. The danger of sudden death should undoubtedly be mentioned to his friends, or to the patient himself. There is, of course, no special treatment other than "expectant" to be adopted, but this palliative treatment is of the greatest importance.

The most concise account of this affection of the aorta is contained in an original article by Dr. Peacock in the "British and Foreign Medico-Chirurgical Review" for April, 1870. Dr. Peacock gives a brief abstract of all the cases recorded up to that time, with full references to the authors.

Rokitansky is the principal authority on the subject. "Path. Anatomy," vol. iv., and his work "Ueber einige der wichtigsten Krankheiten der Arterien," 1852.

See also section on "Coarctation of the Aorta" in Dr. Walshe's work on "Diseases of the Heart."

In Foerster's "Handbuch der Pathologischen Anatomie," p. 726, will be found a brief description, with references to fifty-two cases by different authors.

ANEURISM OF THE ABDOMINAL AORTA.

BY DR. WILLIAM MURRAY, F.R.C.P. LOND.

THE pathological anatomy of Aneurism, the degeneration of arteri
and the process of erosion in bony structures, are subjects too gener
to be handled in an article on the diseases of a single blood-vesse
the following remarks will not therefore be so extended as to embra
those general conditions to which the aorta is subject in common wi
the whole arterial system. These will be alluded to when they prese
features peculiar to the abdominal aorta; but the discussion of t
several diseases to which this lower half of the vessel is subject w
chiefly engage our attention. The latter afford so deep a study
pathology, diagnosis, and treatment, that a distinct treatise might w
be written on them, especially as they often involve excruciati
agony to the sufferer, and have in most cases a fatal issue.

THE ANATOMICAL CHARACTERS OF THE DISEASE.—Atheromato
calcareous, or ossific (Virchow) changes in the coats of the aorta
doubtless the chief predisposing causes of Aneurism; they rob t
vessel of its elasticity by destroying its middle coat, where resides t
tissue on which the dilatation of the artery and its subsequent rec
depend, and thus they lead either to a permanently dilated conditi
of all the coats of the vessel (at the diseased spot), constituting a *tr*
Aneurism, or the degenerated coats give way and a *false Aneurism*
produced. In addition to this source of origin, a tolerably healt
aorta may be ruptured in its middle or internal coats, and thencefor
an aneurismal pouch may be formed at the seat of the accident; a
this fact is worthy of remembrance, because an Abdominal Aneuri
arising from muscular exertion or external violence may thus occur
a healthy vessel, and be subjected to successful treatment, whereas t
occurrence of the disease apart from straining or violence, points stron
to a diseased condition of the vessel, and augurs ill for the future cou
of the disease. It is also important to remember that Abdomi
Aneurism is much less often complicated by valvular disease of t
heart or extensive arterial disease, than is found to be the case
Aneurism of the thoracic aorta and its branches.

Simple dilatation of the abdominal aorta is rare, and the cases
true Aneurism bear a small proportion to the cases of false Aneuri
in this vessel. Dr. Sibson's tables show 60 per cent. of Aneurisms

the abdominal aorta to have been sacculated and 1 per cent. non-sacculated. If we may take it for granted that almost all the sacculated Aneurisms were false, and that some of the non-sacculated were also false, we see how small the number of true Aneurisms in this situation becomes. The opening into the Aneurism may be at any part of the circumference of the aorta. In sacculated Aneurisms it is as frequently at the anterior as on the posterior aspect of the vessel, and it may be exceedingly small, or as extensive as the size of the vessel will allow; in one case the whole of the posterior aspect of the vessel had disappeared. The coats of the artery in true Aneurism are, of course, continuous throughout; but when, as does occur, the true Aneurism is sacculated, the inner coats become extremely delicate, and the external coat becomes thick and strong. If the inner coats become thickened they are never thereby increased in strength or tenacity. In the cases I have examined when the Aneurism was sacculated and of moderate size the internal and middle coats were prolonged but a short distance into the sac; here they became soft and pultaceous, or rough and adherent to the fibrine or other contents of the sac, and in tracing them throughout the sac fragments were here and there discernible in patches. The sac may be empty, or merely lined by a thin layer of fibrine when the Aneurism is small and communicates with the artery by a large orifice so as to permit a free current of blood through the cavity of the sac. In other cases, where the current has not been so free, concentric laminæ of fibrine are found, tough and old immediately beneath the external wall, but softer and stained with blood towards the interior of the sac. The sac may contain coagulated blood in quantities varying according to circumstances, and some have observed a distinct vascularity in the external layers of fibrine. I would lay particular stress upon the presence of *laminated fibrine and coagulated blood* in these Aneurisms, because one or other of these in any given case is the chief factor in curing the disease; they are therefore to be looked upon as the result of nature's unaided attempts to provide a means of cure. In some rare instances the Aneurism has been formed by a hernial pouch of the internal coats protruding through an aperture in the external coat; in other instances the blood finds its way between the internal and external coats, and thus forms *a dissecting Aneurism*. Laennec saw an aorta in which the internal and external coats were thus separated from the arch to the bifurcation; and a case is quoted in which the blood thus dissected its way for a few inches and then passed into the original channel of the vessel by another opening just above the bifurcation, and thus establishing a fresh course for itself, obliterated the natural channel of the aorta. The natural tendency of the disease is to increase, despite the reparative deposition of barriers of fibrine or coagula; on this account the sac may attain a great size, but in doing so its walls usually give way, and a *diffuse Aneurism* is formed. In some cases the original walls disappear entirely, and their place is taken by adjacent textures without the occurrence of any great extravasation of blood. And thus the walls of an Aneurism may come be formed of fasciæ, bones, viscera, layers of fibrine, &c.

The changes which occur in the sac are either conducive to the cu of the disease or to the occurrence of rupture. The latter is by far tl most common tendency; for, despite the reparative deposition of ba riers of fibrine, the Aneurism increases in size under the pressure of current of blood too rapid to coagulate or deposit fibrine, and eventual the sac gives way, either forming a diffuse Aneurism, or destroyir existence at once by the loss of blood in large quantities, or by su cessive hæmorrhages letting life ebb out more gradually. If a diffu Aneurism be formed, the diffused blood may coagulate, layers of fibri may be deposited, and these, together with adjacent parts, m contribute to prevent further extravasation of blood, so that life m be considerably prolonged; in the end, however, a diffuse Aneurism almost always fatal, either by the enlargement of the tumour or by i rupture. The other termination to which the disease progresses that of cure. Hodgson relates a curious case of a small Aneuris which had eaten its way into the body of one of the dorsal vertebr and had there become completely filled with layers of fibrine, whi presented a smooth even surface to the channel of the aorta. Agai the Aneurism may become so placed as to press upon its aperture communication with the aorta, and thus may lead to its own cure compression. From whatever cause the deposition of fibrine arises will lead to the safest cure of the disease, but generally the filling of the sac will be aided by coagulation of blood between or within t layers of fibrine. The sac may become obliterated by inflammati and suppuration of its walls and contents. Haldane reports a case which three Abdominal Aneurisms underwent spontaneous cure calcareous degeneration of fibrine which had coagulated in th interior.[1]

Seat of the Disease.—We have already said that an Aneurism m commence at any part of the circumference of the aorta, and I Sibson shows that it occurs just as frequently on the anterior as the posterior aspect of the vessel. Generally the tumour inclin to the left side, but in some cases it has been seen projecting acr the front of the spine towards the right side. It may be seat beneath the pillars of the diaphragm, and being embraced by the may project into the chest as well as the abdomen. Most frequent the disease occurs near the origin of the cœliac axis, and it often i volves the orifice of that vessel; the origin of the superior mesente artery is also a common seat—indeed, it may be said with truth th the disease usually occurs above the renal arteries. I *have* seen seve cases, however, in which the disease was seated below the renal vesse and one in which it must have been as low as the origin of the infer mesenteric artery. As a rule, the arteries near the Aneurism beco involved in it, and sometimes they are dilated; when the Aneurism large they are often stretched over it, and in most of these cases they obliterated either by pressure or coagulation of fibrine. Pressure upon other parts occurs as the Aneurism enlarges; it may become doubled on the aorta, and thus compress that vessel itself; or, as already observed, the various branches of the aorta may be compressed and obliterated—compression of the bile-ducts may occur, leading to jaundice; of the duodenum and pylorus, causing nausea; of the cardia and œsophagus, producing vomiting and dysphagia; or of the renal vessels (generally without uræmia, or suppression of urine). It is curious that compression of the renal vessels so seldom leads to serious results. I have a specimen in which the left renal artery is obliterated, and the corresponding kidney is dwindled to one-third its natural size, while the artery of the opposite side is pervious, and its kidney is hypertrophied to double its natural size.[1] In this case no renal disorder manifested itself by symptoms during life. I do not know of any proof that the thoracic duct suffers from compression in the abdomen, although such a complication with consequent emaciation is highly probable, nor have I heard of the *pancreatic duct alone* suffering in a similar manner. The left kidney may be considerably displaced, and pushed over to the right, or the liver may be pushed forward, and the disease may thus simulate enlargement of that organ. The vena cava is so far removed from the aorta above the level of the renal arteries, that it is seldom compressed; and dropsical accumulations, or enlargement of the superficial veins, are on this account rare in Abdominal Aneurism; lastly, the colon may be obstructed in its descending division with the occurrence of symptoms of obstruction during life.

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[1] Edinburgh Medical Journal, January, 1863.

The aneurismal sac will protrude anteriorly, forming a considerable pulsating tumour, if it spring from the anterior aspect of the vessel; if, on the other hand, it spring from the posterior aspect of the vessel, it will be bound down by fasciæ or other structures and protrude but little; while, however, these posterior Aneurisms pulsate and protrude but little, they lead to more serious results by pressing on important deep-seated parts. As the nervous pains which form the chief symptoms of Aneurism are the result of this pressure, it may be well to indicate the anatomical relation of anterior and posterior Aneurisms (*i.e.* Aneurisms springing from the anterior and posterior aspect of the aorta) to the nerves in which the pain is chiefly seated. An anterior Aneurism, which springs from the anterior aspect of the aorta and protrudes forwards, will necessarily compress the ganglia plexuses and branches of the abdominal sympathetic system; a posterior Aneurism, which springs from the posterior aspect of the aorta and grows in a posterior direction, presses on the roots or branches of the spinal nerves as they issue from the intervertebral foramina, in close proximity to which the aorta is placed at this part of its course. Corresponding with these anatomical facts, Dr. Sibson has shown the rule with regard to these aneurismal pains to be: that posterior Aneurisms excite paroxysmal and radiating, as well as continuous pains, in the back and loins in a large number of cases, and pains in the epigastrium in small number of cases; while anterior Aneurisms excite pain in th epigastrium in a large number of cases, and pain in the loins wit paroxysms of radiating pains in but a small number of cases. Thu there is established a direct relation between the situation of th Aneurism and the seat of the pains produced by the disease, and thi is fairly explicable by the fact that *anterior* Aneurisms press on th *sympathetic* nerves of the abdomen, while *posterior* Aneurisms press o the *spinal* nerves and their branches. All this is said with a fu recognition of the fact that erosion of the vertebræ is produced fre quently by posterior Aneurisms, and but rarely by anterior developments of the disease—for the truth is, that no relation whatever ca be established between the pain of these Aneurisms and the occurrenc of erosion. Sibson found, we admit, that of forty-six cases of sacculated Aneurism, in fifteen there was erosion of the vertebræ, and i every one of these fifteen there was a communication with the aort on its posterior aspect; but Habershon and others have clearl established the fact, that as to cause and effect no relation betwee pain and erosion of the vertebræ exists for cases are recorded in whic there was pain in the loins of the most acute nature, and after deat the vertebræ were found to be free from erosion; and a painles illness has more than once been known to precede death when th vertebræ were found to be extensively eroded. The conclusions to b drawn are these:—1st. That sacculated posterior Aneurisms, as a rul produce erosion of the vertebræ, and are generally accompanied b lumbar pain, but cases may occur where, by pressing on the spina nerves alone, pain without erosion may be produced; and conversel sacculated posterior Aneurisms may in rare cases erode the vertebr without pressing on the spinal nerves, and therefore without producin pain: in short, you may have pain without erosion, and erosion withou pain, for the pain depends upon pressure on the spinal nerves, *and n upon erosion.*

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[1] See a specimen, in Path. Division of the Newcastle Museum.

Erosion of the vertebræ occurs chiefly in Aneurisms which open int the aorta on its posterior aspect. By this process the bodies of th vertebræ may be completely destroyed, and then the Aneurism ma communicate with the spinal canal, or the vertebral column may b considerably displaced, and angular curvature may be thus produce In a case already referred to, the body of one of the lumbar vertebr was completely hollowed out by a small Aneurism, and the cavity thu formed was lined by a fine smooth membrane. I need scarcely sa that erosion of bones is a process distinct from caries, consisting of combination of absorption and molecular destruction under the influence of pressure. *True caries* of the vertebræ has been observed i connexion with aneurismal pressure, and this is not at all to be wondered at, if the aneurism occur in a strumous subject, or if the bon has previously been in an unhealthy condition.

The rupture of Abdominal Aneurisms is more frequently followe by the formation of a *diffuse Aneurism* than is the rupture of an

[illegible]

[illegible]

Habershon, Stokes and others have [illegible] rupture [illegible] the aorta [illegible] in which case the blood may find its way upwards into the chest [illegible] as Stokes says, a [illegible] of blood [illegible] and Dr Beatty record cases of rupture into the spinal canal. [illegible] may burst into the lungs [illegible] happened where [illegible] adhesions [illegible] part of the parietes of the Aneurism [illegible] found its way to the [illegible]. Lastly, rupture into the vena cava is mentioned [illegible] cases. Mr Syme's case of varicose Aneurism between the aorta and the vena cava is, as far as I know, unique.

THE CAUSATION OR ÆTIOLOGY OF THE DISEASE.—The chief predisposing cause of Aneurism, here as elsewhere, is degeneration of the arterial coats, but this is not the sole cause, as the aorta in the abdomen, though much less frequently degenerated than the [illegible] aorta, is nevertheless as frequently the seat of Aneurism as that vessel. This proclivity to Aneurism is due, no doubt, to the position occupied by the abdominal aorta: closely bound to the spinal column in its most mobile part, it is subjected to every position the body may assume—at one time being greatly on the stretch, at another [illegible] itself—nor are the variety and extent of the movements to which th vessel is subjected the sole causes of this disease, for the rapidity wit which they are suddenly performed is often of itself sufficient t lacerate its brittle inner coats. This latter cause is seen to act i cases where the patient has suddenly attempted to regain his balanc or where he has made a sudden start or effort, from which he is ofte able to date the commencement of his malady.

We may, therefore, lay it down that muscular exertion is the chi exciting cause of the disease, and that this may operate in two ways 1st. By suddenly subjecting the vessel to a severe strain. 2nd. B continually subjecting it to a variety of movements which increase t strain upon its internal coats. Fully according with these statemen we find that intemperance, severe privations, advancing age, irreg larities of life, and dissipation, syphilis, and rheumatism (all of whi induce gradual degeneration of tissue), are frequently adduced predisposing causes of the disease. We find that occupations a conditions of life leading to great muscular exertion are prolific exciting causes of the disease; for instance, of 49 cases, Crisp fou that 47 were between twenty and fifty years of age, and that 22 these were between thirty and forty years of age; so that, while a undoubtedly contributes to produce the disease, it is not *old age* esp cially, but a certain time of life (when degeneration may have set i which is generally accompanied by great bodily activity. In short, small amount of degeneration accompanied by great bodily exerti more easily leads to the disease than a large amount of degenerati with slow and feeble bodily exertion. Corresponding proof of t is found in the frequency of the disease in males as compared wi females. Crisp found 8 female to 51 male cases; Habershon, females to 11 males. As a rule, when the disease occurs in femal they are young, and have led very hard and irregular lives of dis pation. Gairdner mentions such a case in a young woman aged o sixteen years. The occupations which seem to predispose to t disease are all of a laborious nature; the men are smiths, strike excavators, navvies, porters, paviours, founders, &c. The disease *m* occur apart from these exciting causes, and when it does so we m be sure there is more certainty of the aorta being extremely diseas and far less chance of the disease being cured than when it is caus by muscular exertion, &c.

In this category of causes we ought to include external injuri such as blows, by which the disease is not unfrequently produce and we cannot lay too great stress upon the bad effects of ov straining, such as the following.

A healthy young fellow is employed as a paviour, and on lifting huge wooden rammer for a blow loses his balance; with the ramm uplifted he makes a vigorous effort to keep his feet, during which "strains his back;" from that day he begins to complain of sympto of Aneurism, and eventually he turns out to have the disease.

SYMPTOMS.—I propose to discuss the symptomatology of the disease under two heads.

1st. Those symptoms and signs which by their presence indicate the disease. *These are the positive symptoms.*

2d. Those symptoms which by their presence *contra*-indicate or negative the existence of Aneurism, and by *their absence*, in doubtful cases, are therefore evidence in favour of the existence of Aneurism. *These are the negative symptoms,* and are of course produced by other diseases which closely resemble Aneurism in their main characters; when these symptoms positively present themselves, they often exclude the possibility of Aneurism; when they are altogether *absent*, we have an ominous sign that the disease exists.

1st. *The positive symptoms.* Pain is generally the earliest symptom, the most prominent symptom during the course of the disease, and the symptom which by its increasing intensity gives the severest sting to the patient's dying moments. With regard to its frequency, we may say it occurs in five-sixths, if not in seven-eighths of the cases on record; its frequency and its general characters are chiefly determined by the seat of the Aneurism.

The absence of pain in some cases is, however, a well-established fact, and when this is combined with an absence of other leading symptoms, the patient may live, till within a few hours of his death, without the slightest suspicion that any serious malady exists. Such a case has occurred to my knowledge where rupture and loss of blood were the cause of death. We have before demonstrated the relation which exists beween the seat of pain and the seat of the disease, and have shown that pain is a more severe and more frequent occurrence in posterior than in anterior Aneurisms. As the nature of these aneurismal pains is peculiar and characteristic, their consideration is of great importance. They are of two kinds. 1st. *A continued pain* in the back, loins, epigastrium, or hypogastrium, as the case may be. 2d. *An intense paroxysmal pain* in the back or loins, which radiates to the front of the belly, to the testicle, or down the thigh, according to the spinal nerves through which it may be produced. The first is wearisome, and exhausts the patient by depriving him of his rest; the second is agonizing, and leaves him prostrate after every paroxysm. The pain is unaccompanied by febrile excitement, acceleration of the pulse, rigors, or perspirations; it usually increases as the disease progresses, and before death agony of the most acute nature has often to be endured: in one case this was so severe that an exhausted and dying patient fairly leaped out of his bed, and in another case it brought on an attack of raving delirium.

We conclude that the rule with regard to pain in Abdominal Aneurism is, that it may be seated in the back or in the belly, indicating in the former site a posterior Aneurism, and in the latter an anterior Aneurism, and that in either case it may be of two kinds —paroxysmal or continued. The situation of the pain occasionally varies: it may be seated in the hip, in the iliac regions, changing from one side to the other (Beatty's case); it may simulate colic, bein increased after eating, or by constipation of the bowels, and may b accompanied by nausea, anorexia, vomiting, or flatulence. The regio of the liver may be painful and even tender, with pain about th shoulder and scapula, thus simulating hepatic disease; the chest ma be the seat of pain with dyspnœa, or the cardiac region with palpitation. One other strong feature of the pain is its tendency to "catc the breath" during the descent of the diaphragm, and in such case we may suspect the seat of the disease to be underneath the crur of the diaphragm.[1]

The paroxysmal pain may closely simulate that of renal or biliar calculus, lead colic, or simple neuralgia.

All these somewhat anomalous pains may seriously complicate th diagnosis, and in such cases a careful consideration of other symptom will be necessary. Change of posture often affects the pain cons derably. Patients usually experience relief by lying on their fac or on their right side, by resting on the hands and knees, or by sittin almost double. On the other hand, lying on the back, or standing i the erect posture, are usually painful positions. The pain is almo always increased by bodily exertion, and in some cases even th slightest movement is attended by pain; pressure usually increase it, but in some instances pressure has afforded considerable relie sometimes the surface over the Aneurism is exquisitely tender; bu all I have seen were quite free from this, and might be manipulate gently with the greatest freedom from pain.

The pain varies in *character* as well as in situation; the wor boring, burning, catching, pulsating, twisting, lancinating, are use by patients to convey to our minds the different kinds of pain fe by them. It is often dull pain when continuous, and sharp whe paroxysmal. It varies in *intensity* also,—it is sometimes absent, other times trivial; in most cases very severe, and occasional unbearable. We would urge the great necessity of recognising th import of pain such as is here described in all obscure cases, ar of making it an incentive to a most careful physical examination the abdomen, in order to determine whether or not an Aneuris exist.

A *pulsating tumour* forms another most important symptom this disease. The pulsation may be visible even at a distance, formi a distinct heaving projection of the abdominal wall; on the oth hand, it sometimes gives no visible evidence of its existence.

To the applied hand a distinct heaving impulse is communicate and at the spot a more or less defined tumour can often be felt. T character of the pulsation is very marked; felt equally in all dire tions—laterally as well as in front—the tumour seems with ea pulsation to expand under the hand, and when the fingers are appli to its sides they are separated with each pulsation. In some case where the sac has pressed on the cardiac region, a double shock pulsation has been communicated to it from the heart. There may be a thrill or vibration with each pulsation, and if by pressure the sac can be emptied, its distension will be accompanied by a purring thrill. The tumour may be moveable when it occupies one of the branches of the aorta, *e.g.* the sup. mesenteric; in opposition to this, however, we have cases by Courato and others, diagnosed to be Aneurisms of this vessel by their non-mobility along with the diaphragm: most frequently the tumour is fixed. The site of the pulsation varies considerably; most frequently it occupies the epigastric region and inclines to the side, so as to be covered by the margins of the left false ribs; in rare cases it inclines to the right side, and in so doing may push forward the right lobe of the liver with each impulse; it not unfrequently occupies a spot a little above and to the left of the umbilicus, or it may be so deeply seated in the epigastrium as only to be felt on deep pressure in that region. The mass may present itself in the left lumbar region, or in the groin, simulating lumbar abscess in the one case, and psoas abscess in the other; or it may project considerably towards the right lumbar region, and so simulate enlargement of the kidney. I have read of one case in which the tumour occupied the position of the spleen, and was mistaken for disease of that organ; and in another case, under my care, whenever the patient lay upon his left side the tumour fell under his left ribs and entirely disappeared. Added to the difficulties which may arise from the various situations of the tumour, is the established fact that, in some cases, neither pulsation nor tumour can be detected. Hope mentions a remarkable case of this kind, where the Aneurism was found to be bound down by the pillars of the diaphragm, the lumbar fascia, and bands of adhesion—an occurrence by no means impossible when the Aneurism is small and projects backwards from the posterior surface of the aorta. As before mentioned, the tumour may sometimes be emptied by pressure, but this will depend upon the amount of fibrine in its interior.

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[1] See cases by Dr. Ogle, in Lancet.

The Physical Characters of the Tumour.—The tumour may be soft and fluctuating, and in such cases it is easily emptied of its contents by pressure, or it may be dense and resistant to the touch; its surface is generally smooth, although in rare cases it may be lobulated. Percussion does not often afford valuable evidence of the disease, for the presence of intestinal flatus, and the deep situation occupied by the tumour when it is above the renal arteries, interfere with its indications. The percussion note is generally dull over the tumour, but this dulness may be limited to one part, such as the summit of the tumour, and there is generally a line of resonance running between the tumour and the liver. The characteristic expansile pulsation of the tumour may be replaced by a forcible short or jogging shock communicated to the applied hand.

A bellows murmur is frequently to be heard in connection with Abdominal Aneurism. It was observed in 25 per cent. of the cases collected by Sibson, but I cannot help thinking that it occurs more

frequently than this, for I have never seen a case without it. T
character of the murmur varies; according to Dr. Walshe it may
1. A single systolic murmur. 2. A dull, muffled, systolic sou
convertible into a murmur by a little pressure. 3. A sharp, abru
systolic murmur, audible at the left lumbar spine, or much m
marked there than in front. 4. A systolic murmur below the s
and none immediately over it. 5. Occasionally a dull second sou
Dr. Walshe has never heard a murmur diastolic in time.[1] A sh
may be perceived by the ear, as well as a murmur; it may be sin
and systolic, or double; the presence of a second diastolic shock
pathognomonic of the disease; as before said, a thrill may accompa
the murmur. Where murmur is inaudible, pressure with the stethosc
over the Aneurism may produce it, and in all cases the patient sho
be made to assume the recumbent position, which generally increa
both the murmur and the pulsation of the tumour. Dr. Corri
attributes this development of the murmur and pulsation in recu
bency, and their disappearance in the erect posture, to the removal
hydrostatic pressure from the walls of the cyst, which permits a m
free passage of blood in and out of it, with less tension and m
vibration of its walls. The murmur varies in intensity: it may
feeble, or so loud as to be audible at a distance, and its intens
sometimes diminishes with the growth of the Aneurism.

It is very important to bear in mind, in obscure cases, that
physical signs may be absent—impulse, murmur, and dulness; and
such cases a diagnosis of the exact nature of the tumour can
possibly be made. Murmur is sometimes the only physical evide
of the disease. In all suspected cases, therefore, the stethosc
ought to be applied, and its application to the spine and left verte
groove ought not to be neglected, as murmur in that region is
grave import.

To these, the cardinal symptoms of the disease, we must add ot
of less importance. *Respiration*, if the sac be so high or bu
as to interfere with phrenic action, will be quickened and impe
Sibson found dyspnœa mentioned in eight per cent. of the ca
Cough may be excited when the walls of the thorax become in
way connected with the sac, but, as this is rare, we find cough m
less frequently present than dyspnœa. *Dysphagia* was observed
about six per cent.; and from the close anatomical relation which
œsophagus bears to the aorta we need not be surprised at t
Nausea, vomiting, loss of appetite, flatulence with spasmodic p
and constipation, are all common enough occurrences in the cours
the disease. Mayo observed numbness of the lower limbs; and
other cases, coldness, formication, pricking, numbness, and e
paralysis of the legs has occurred. Ascites or anasarca of either l
are exceedingly rare, and, as will be seen, these occurrences are n
indicative of the absence of the disease than of its presence, *i.e.* t
are negative symptoms which when present almost preclude

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[1] Diseases of the Heart, p. 494, 3rd ed.

existence of the disease. As the result of pressure, we ought to mention Professor Seaton's curious instance of contraction of the right pupil in a case of Abdominal Aneurism; probably the sympathetic nerves were the channel through which this curious phenomenon was brought about.

It is a curious fact that all these symptoms may for a time disappear, and the patient get apparently well; it is curious, too, that during seasons of great excitement, when the mind is directed to other things, the pains may be completely forgotten. The cases of the gentleman who spent a day in the hunting-field, and the barrister who made a powerful speech in court, a short time before death, abundantly illustrate this. As the disease progresses and the pain increases, insomnia and restlessness at night tend to exhaust the body; the patient becomes emaciated, exhausted, worn out, and would doubtless die of slow exhaustion did not death from rupture put an end to his sufferings.

2d. We now come to *the negative symptoms,* by which we mean those symptoms which seldom or never occur in Aneurism, and therefore negative the existence of the disease; these symptoms being generally present in diseases which simulate Aneurism, are of great importance in the diagnosis of doubtful cases. They speak against the existence of Aneurism, and for the existence of other diseases.

We first remark the absence of general arterial excitement, with undue fulness or rapidity of the pulse—78 to 88 seems to be the average rate of the pulse, and the pulse-respiration ratio generally remains at the normal standard. Undue rapidity of pulse is therefore a negative symptom. The temperature of the body is, as far as I have observed in two cases, normal, and from the exhausted state of the patients, it may be below par; great increase of temperature indicates some other affection. The heart's action may continue to be quite normal; indeed, we may say disease of the heart is the exception rather than the rule in this disease.

Anasarca of the extremities, so important in many abdominal diseases, *is very rare* in Aneurism, and only occurs when the cava becomes involved in or communicates with the tumour.

Enlargement of the superficial veins of the abdomen is also exceedingly rare in Aneurism, and very common in other diseases of the abdomen. Ascites is still more rare, and being so common in abdominal disease, its absence is seriously significant of Aneurism. Dr. Stokes lays great stress upon the absence of effused lymph and the friction-sound which it produces when it is present in the peritoneal cavity Unless the disease press on the emulgent vein of the kidney the urine is healthy; and as, clinically, very few exceptions to this rule occur, the absence of sediment, of pus, mucus, and albumen, ought to prevent us from assigning a renal or vesical origin to the pain. Jaundice, want of bile in the stools, general constitutional disturbance with complication of other organs, and enlargement of the liver, are opposed to the probability of the existence of Aneurism.

Mobility of the tumour rarely occurs in aneurism of the aorta its although occasionally this is observed in Aneurism of its branch but mobility is, as we shall see, common enough in some other ab minal tumours. Stokes points out that aneurismal tumour ne begins below and increases upwards ; this upward growth of a tum would therefore argue powerfully against Aneurism.

The general cachexia of cancer, or the tubercular diathesis, are v rare in Aneurism, as are also deposits of cancer or tubercle ; so that all doubtful cases, careful inquiry into the history of the patient, careful physical examination of the body, are necessary.

The diseases which may be mistaken for Aneurism are so numer and diversified that it may be well to divide them into—

1. Affections depending on increased aortic pulsations only.
2. Affections wherein a tumour, itself pulseless, receives an imp from the aorta.
3. Affections wherein pulsation, not derived from the aorta, exist
4. Affections wherein a pulseless tumour simulates a non-pulsat Aneurism.
5. Affections where pain without tumour exists.

1. Increased *Aortic pulsation* is not expansile, the lateral imp and the movement in a forward and slightly downward direction be wanting. Murmur is very rarely present (never occurring over spine, and scarcely ever diastolic), and the most careful manipulat and percussion will fail to detect lateral enlargement in calibre. absence of pain, of the evidence of arterial disease, and the oc rence of the disease in young, anæmic, and female subjects of nerv temperament, or its development during attacks of dyspepsia, fl lence, or constipation, and its entire removal by treatment, are dications sufficient for our guidance in ordinary cases. In obscure cases, the most careful inquiry will often fail to produ decided opinion.

2. Tumours which receive an impulse from the aorta.

Under this head are cancer of the stomach and pylorus; canc the pancreas, liver, and small omentum, involving the duode Tubercular deposits in the mesenteric or lumbar glands; ome tumours or cysts, ovarian tumours, and tumours attached to the ut or even the enlarged uterus itself; enlarged kidney, distension of pelvis of the kidney, a moveable kidney, distension of the colon flatus, worms, or fæces (the latter being by far the most commo cancer of the transverse colon, each of these affections may rec an impulse from the aorta, and may compress the vessel so as to to murmur also. By attention to the symptoms before deline many of these may be excluded; and, as the diagnosis in each case require a full consideration of each suspected disease, all I shall att to do here will be to lay down a few general rules for our guidanc

A. In but few of these cases does the impulse possess the hea expansile character of Aneurism; and lateral impulse is rare.

B. The tumour in most of the above cases is moveable, and moved the murmur is modified thereby; in Aneurism of the Aorta itself this is never the case; and in Aneurism of its branches, movement does not cause the murmur to disappear; by causing the patient to rest on his hands and knees the tumour may fall away from the aorta, and pulsation will then cease if it be not aneurismal.

C. In many cases symptoms contra-indicating Aneurism will be present.

D. In all cases a more careful inquiry into the history of the case, the constitution of the patient, and the character of the tumour, is necessary, for in many cases this will give great aid to diagnosis.

E. Clear out the bowels, examine the urine, remove dyspeptic symptoms, regulate the uterine functions, and in so doing make careful inquiry into the condition of the organs involved.

3. This class of cases includes those tumours which possess in themselves a heaving impulse, due to their active vascular condition. The relation between a diffuse Aneurism and a vascular cancer is sometimes very close, even when external; and when vascular organs like the liver or kidney becoming affected with hæmatoid cancer, acquire a heaving, expansive pulsation, the diagnosis is very difficult. Cancer of the mesenteric and lumbar glands may also simulate Aneurism. It is in these cases that the presence of constitutional disturbance, quick pulse, hot skin, perspiration, and of diathesis, is so useful an aid to diagnosis; the symptoms of disease of the organs involved would also aid us if present.

4. Cases where a pulseless tumour resembles a non-pulsating Aneurism.

Of this class are psoas and lumbar abscess, possibly leading to displacement of vertebræ, like angular curvature, causing pain in the back like that of Aneurism, and perhaps appearing suddenly as if the Aneurism had ruptured. We must include in this class any of the tumours in class 2 which may have acquired the symptoms of Aneurism without pulsation. The absence of murmur in these cases, and the presence of cancer or tubercle in other organs, would aid the diagnosis.

The 5th class of cases is a difficult one. It includes all cases of obscure pain in the belly, back, loins, hips and thighs. Cases of lumbago, sciatica, neuralgia of the abdominal wall, intestinal pain, the pain of calculus, various hepatic and nephritic pains, spinal pain, &c., &c. In such cases a careful physical examination of the abdomen, for other evidence of the disease, is the best safeguard in avoiding error; but beyond this, and the special symptoms of these diseases which may accompany the pain, I know of no point on which to establish a diagnosis.

Course, Duration, and Termination of the disease.—Beginning suddenly, or developing gradually by obscure symptoms, the course taken by the disease varies; it may be so mild as never to betray its existence till the final rupture takes place, or it may entail almost constant suffering on its victim. In pronouncing an opinion it is of great

importance to remember that even in severe cases periods of conv
escence occur in which all symptoms disappear, and the patient fe
quite well. Many cases seem to exist for a long time without serio
disturbance, when suddenly, after the Aneurism has reached a certa
size, all the symptoms set in with great severity. The duration of t
disease is variable; cases are on record which lasted seven years, a
even eight years, but this is far above the average. The practi
question seems to be, how long will a patient live after an Aneuri
has produced severe or decided symptoms of its existence? T
period rarely exceeds eighteen months, and after the formation o
decided pulsating tumour, with paroxysms of pain, the majority
patients die within three months. The termination of the disease
hitherto, in almost all cases, been in death. A few cases are recor
where, without aid, nature managed to fill up the sac by layers
fibrin, &c., and thus to establish a cure. In other cases again,
proper diet and regimen, and the employment of various modes
treatment, the disease has been cured.

Death may occur from exhaustion, or from hæmorrhage; and de
from hæmorrhage may be sudden or gradual. When gradual,
patient may at first experience relief of his pain, or shock with rig
may be experienced at the time, and last more or less for a few da
during which the heart beats rapidly and feebly, the first sound be
almost lost. The pulsation in the tumour, after rupture, beco
weaker, the murmur is lessened, and a new, soft, semi-solid swell
is formed without pulsation or murmur. Dr. Walshe has obser
pulsation of expansile character in the extravasated blood, but the
is, I believe, as stated above. Dr. Lyons found the symptoms of r
ture followed by dulness on one side of the chest, and diagno
rupture into the pleural cavity, which proved to be correct. Hæm
rhage into the peritoneal cavity is generally followed by sudden de
and hæmorrhage into the lung, stomach, or bowel, will be followed
discharge of blood from those cavities. Convulsion may accomp
loss of blood. Dr. Stokes thinks that a sudden loss of blood is m
dangerous than the loss of larger quantities by degrees. In one c
Dr. Stokes observed fainting on three successive days before dea
he believed them to correspond to successive discharges of blood f
the Aneurism. When the blood forms a tumour in the lumbar or i
region, the pulsations in the femoral may be impeded, but this is
always the case.

TREATMENT.—Like most uncured diseases, Aneurism of the A
has been subjected to various methods of treatment. It will be need
to dwell upon those methods which have become obsolete, and h
been rejected by the experience of the profession. Of these the tr
ment of Valsalva has been most commonly adopted, for in most c
it affords temporary relief, and in some cases seems to have establis
a cure, where the strength of the patient, aided by his faith and
severance in the means employed, permitted of a fair trial. We sh

scarcely have adverted to the plan of Valsalva, had not Tufnell, of Dublin, brought out some rather startling cases showing that a modification of Valsalva's treatment may prove successful. Dr. Tufnell's plan is that of Valsalva without depletion, and consists chiefly in rest, plain but good diet, and soothing medicines when needed. The patient should be placed in the recumbent position, and this he must maintain for some weeks, or even months. Now, despite the success which has attended Dr. Tufnell's efforts, I cannot think his treatment will ever become popular, for few patients will be found willing or able to rest night and day in one posture for a period of three months, and the irksomeness of the patient's own existence during that time is not the only difficulty, for the patience of relatives will also be severely tested in giving all due attention to the case; however, when no better means can be employed this may be tried. The administration of medicines has hitherto done but little for internal Aneurism, and further researches in this respect are needed. Dr. Owen Rees has given acetate of lead, and Roberts, Balfour, Begbie, and others have shown how decidedly the symptoms of internal Aneurism improve on the administration of large doses of iodide of potassium. In my own experience the iodide has, in some cases, relieved the symptoms; in others, the symptoms have altogether disappeared for a time, and in one case I believe a cure has been effected. This experience, I think, fairly represents that of the profession generally on this subject. When other means of cure fail, or when they are inappropriate, there still remains one procedure which holds out a fair hope of recovery to the patient. *Compression*, which has done so much for the cure of Aneurism of other arteries, has acquired a peculiar honour in proving applicable to Aneurism of the Abdominal Aorta; in other arteries it supersedes another less safe, but equally effectual mode of cure (ligature); but in the aorta it renders curable a disease hitherto entirely beyond our reach. As a student in some of our best hospitals, I never heard so much as a hint that efforts for the cure of such cases were worth entertaining. Now, however, there is hope for many cases. Already the aorta has been successfully compressed several times for the cure of Aneurism—both for Aneurism in its own course, and for iliac Aneurism. As this method of treatment, though easy in its application, requires much care and attention to details, I propose to add as much information on the subject as we at present possess, in the hope that ere long our experience of the treatment will be much enlarged.

Let us premise by a few remarks on the anatomy of the abdominal aorta. The vessel extends from the last dorsal vertebra to the middle of the fourth lumbar vertebra; it lies a little to the left of the median line, having the right crus of the diaphragm, the vena azygos and the thoracic duct between it and the vena cava. The vena cava is nowhere in exact contact with the aorta, and gradually diverging as it ascends, at the level of the renal arteries the greater part of an inch intervenes between it and the end of the aorta. During part of its course the aorta is embraced by the pillars of the diaphragm, so tha for about two inches only part of its circumference, the anterior aspec is in the abdominal cavity; this embraced part of the artery is remarkable for being covered by important organs, and for giving off the mos important branches of the aorta. It is covered by the pancreas an the splenic vein, the left renal vein, the third portion of the duodenum and some of the most important plexuses of the sympathetic nervou system.

The arteries given off from it are placed as follows:—1. The phreni —immediately the aorta appears from beneath the diaphragm. 2. Th cœliac axis—one inch from the commencement of the abdomina aorta. 3. The superior mesenteric—three-fourths of an inch belo the cœliac axis or one inch and three-quarters from the commencement of the aorta; and the renal arteries, half an inch lower than th mesenteric, or two inches and a quarter from the point at which th aorta enters the abdomen.

From this point downwards for three inches at least, no large vess arises from the aorta, nor is the vessel, during this part of its cours covered by important parts.

Lastly, we have the inferior mesenteric artery, a little more tha five inches below the commencement of the aorta, the vessel itse terminating at the distance of seven inches from its origin.

From these facts it will be seen that the aorta may be compresse with the greatest safety and facility in its lower two-thirds, that is, i the last five inches of its course. Let us refer for a moment to th points on the front of the abdomen, to which this and other poin correspond, so that we may determine from the external evidence the site of an Aneurism, how far it is amenable to treatment b pressure, and with what amount of safety pressure may be applied.

The aorta extends from the end of the sternum to the umbilic (seven inches). Allowing one inch for the length of the ensifor cartilage, its tip will correspond to the origin of the cœliac axis. Thre fourths of an inch lower is the superior mesenteric artery, and half a inch lower than it, or four inches and three-quarters from the umbil cus, is the origin of the renal arteries. Allowing an inch for the poi on which pressure is applied, we have here a clear space of more tha half the aorta where Aneurisms may be easily and safely treated b pressure, for the inferior mesenteric artery being by its anastomos well supplied with blood, can be occluded without the slightest ris The rule is, that you must not apply the tourniquet higher than o inch and a quarter from the tip of the ensiform cartilage, the point origin of the renal arteries; in fact, you cannot apply it higher tha this point, so that we may conclude it is safe to apply it as high you can. For an account of the vessels by which the circulation carried on inside and outside the abdomen after the aorta is occlude I must refer the reader to my work on *The Rapid Cure of Aneuris* and for a still fuller account of the anastomosis between the lumb branches of the aorta above and the branches of the internal iliac belo

I would refer to the researches of Prof. Turner of Edinburgh. It may not be out of place here to add a few words on the application of pressure to the aorta, as its success depends entirely upon an accurate attention to the details of the procedure.

Before proceeding to compress, it is important to ascertain that the heart, liver, kidneys, and other organs are in a healthy state. It is also important to clear out the bowels both by a purgative and by a stimulating enema, as distension of the abdomen by flatus adds to the difficulty and danger of applying firm pressure. The use of chloroform is absolutely essential for more than one reason. It not only enables the patient to bear severe and long-continued pressure, but it also removes the resistance of the abdominal parietes. The instrument to be used may be a large Signorini's horse-shoe tourniquet, or Liston's tourniquet for the abdominal aorta, or a tourniquet with a fine adjustment, invented by Dr. G. Y. Heath and myself, and made by Coxeter. The strength of the rack and pinion, or screw of the instrument, is of very great importance in all cases.

In applying the tourniquet, complete arrest of the circulation through the Aneurism should be aimed at, as it is of the greatest moment to secure complete stagnation of a mass of blood in the Aneurism for a period of time sufficient to produce coagulation. It is very probable that coagulation sets in rather suddenly when once the conditions for it have been secured, and one object should be to encourage this tendency in the blood by retaining it in a stagnant condition as long as possible without the slightest movement or disturbance.

After considering the experience derived from recent cases, I am of opinion that we should proceed as follows in carrying out the pressure treatment:—

1st. Apply the pressure for four hours, and if on removing the tourniquet no impression has been produced on the pulsation, the first attempt must be considered at an end, but if the pulsation is somewhat diminished, the instrument should be reapplied for another hour. In one case when pressure was being applied to the aorta for the cure of an iliac Aneurism, when I arrived I found the assistants who had charge of the case had just removed the instrument in despair, but perceiving a distinct lessening of the pulsation, I insisted on a reapplication of the instrument, and in twenty minutes the Aneurism was consolidated, and the patient got well.

2nd. If the first attempt has been unsuccessful, a few days must elapse before another trial is made; on this occasion the pressure should be maintained for six or eight hours.

3rd. If this fail, a final effort must be made, and the pressure extended to a period of twelve hours. There is but little danger if the process be tried in the above cautious manner, as indications of inflammation, exhaustion, or shock would at once put a stop to our efforts. Pressure so prolonged as to exhaust the patient, or so frequently applied as to prevent perfect recovery in the interval, is specially to be avoided. I need scarcely add that everything depends on tl personal superintendence of the surgeon who undertakes to treat tl case. He must give up his ordinary work for the day, and devot himself to this alone. Assistants cannot be expected to give th concentrated attention to the tourniquet which is essential to th proper application of pressure.

If we fail to cure the Aneurism by any of the above efforts, w must endeavour to alleviate the sufferings of the patient as much possible. Leeching and cupping are sometimes useful in relieving tl pain of plethoric patients. Rest is an all important condition for tl relief of pain. Nocturnal pains and insomnia are best combated l opiates, bromide of potassium and chloral in appropriate doses; bell donna combined with opium is of great use, and liniments of aconi or belladonna and chloroform are useful external applications, but tl subcutaneous injection of morphia is our best remedy for pain. It important to keep the patient's mind free from undue anxiety, and maintain the general health by diet and regimen. Medicines whic control arterial excitement, such as aconite, hydrocyanic acid, gree hellebore, and digitalis, are often of great use, but in spite of the and all other remedies the patient will often suffer severely before l dies.

For much of the pathological anatomy on this subject we a indebted to the labours of Dr. Sibson and Dr. Habershon, and Mr. Timothy Holmes we are indebted for the fullest exposition of tl surgical aspects of the subject, as well as for the first suggestion th pressure might possibly be applied to the abdominal aorta for tl cure of Aneurism. Up to the commencement of 1873 at lea twenty Aneurisms had been treated by pressing the abdomin aorta, of these only two died, and in them the fatal result w evidently brought about by re-applying the tourniquet too soo I cannot too strongly urge the importance of avoiding this; nothing gained by a speedy re-application, and great risk is thereby incurre I need not say that the advocates of a new method of treatme should, above all things, avoid the arrest of its early development a tender growth, by too rudely or hastily putting it to the test inappropriate cases, or by pushing it too far.

Since the above date several cases have occurred. Before referri to them I ought to mention the case cured by Moxon and Durha in Guy's Hospital, and to the case treated by Sir J. Paget witho success. I ought also to refer to the case where Mr. Bryant appli distal pressure with fatal but not altogether discouraging results. full discussion of the treatment used in these cases is contained Mr. Holmes's "Lectures on the Surgical Treatment of Aneurism."

Since 1873 Dr. Headlam Greenhow has successfully carried out tl treatment in a case in the Middlesex Hospital, and Dr. G. H. Philips has succeeded in curing a case in the Newcastle Infirmary. To the might be added one or more cases cured by continental surgeons, a I must not fail to refer to the success of Mapathea and O'Ferral

Dublin in applying pressure to the aorta for the cure of ilio-femoral Aneurisms. Most of the above cases illustrate one important point, viz., that an Aneurism often continues to beat for several hours after the pressure has been removed, and then suddenly and finally ceases to do so. To account for this, several suggestions may be offered. It may depend on a gradual contraction of the walls of the Aneurism, fresh laden with barriers of fibrin; or on the sudden formation of a clot of blood on recently-deposited fibrin, or blood-clot; or on some more obscure alteration of the relationship of the contents of the sac to the blood-current. For a fuller discussion of this interesting question I must again refer you to my work on the rapid pressure treatment.

DISEASES OF ARTERIES.

BY JOHN SYER BRISTOWE, M.D., F.R.C.P.

(1.) *Inflammation.*—Arteritis, or inflammation of the arteries, is disease which physicians and surgeons regarded formerly as of commc occurrence; but they not only included under the term true cases the disease, but attributed to Arteritis cases in which, though arteri disease was present, it was not inflammatory, and probably a sti larger number in which there was no arterial affection whateve They regarded, in fact, mere *post mortem* blood-staining of the linir membrane of the arteries as evidence of inflammatory congestion, ar all degenerative changes of the same tissue as the results of tl deposition of inflammatory products. It need scarcely be said he that the lining membrane of arteries is now known to be devoid capillary vessels, and incapable, therefore, of vascular injection; ar that any redness it may present is simply due to its imbibition aft death of the colouring matter of the blood which has lain in conta with it; and that atheromatous and other degenerative chang have either no connection with inflammation, or only such a coi nection with it as have cirrhotic and such like deposits in the liv and other organs.

Inflammation of an artery manifests itself chiefly by changes in tl outermost and middle, or vascular, coats. In the early stage, tl cellular coat, which is that in which the vasa vasorum are chiefly di tributed, presents changes similar to those which occur in inflamed coi nective tissue of other parts; these are, specially, redness from hype ræmia of the capillary vessels, and swelling in consequence of inflan matory exudation. Similar changes, though less in degree, affect tl outer portion of the middle coat. But the inner layer of the midd coat, and the internal coat, undergo little if any appreciable modific tion. If, however, the thickening of the walls be at all considerabl then the calibre of the vessel becomes diminished, and the linir membrane corrugated. The inflammatory changes may becon arrested at the point here indicated, and the artery then either reve to its original healthy state, or its walls become permanently ind rated and thickened; or, on the other hand, the inflammation ma lead to softening, and even to suppuration—the latter conditic affecting specially the outer portion of the arterial parietes. Tl processes just described as occurring in the arteries of man, have been proved experimentally, by Virchow and other observers, to be capable of production in the arteries of dogs and other of the lower animals.

Associated generally with arteritis, especially with inflammation of arteries less in size than the aorta, we find the deposition of fibrin on the inner surface of the inflamed tract. This soon increases in quantity, and occludes the channel of the artery. A fibrinous concretion is thus produced, adherent to the surface, blocking up the vessel, sometimes more or less defined and rounded at the extremities, sometimes shading off into ordinary undecolourized coagulum. After a while such a clot is apt to undergo disintegration in its interior, to become a cyst occupied by puriform fluid, and thus to resemble accurately the softening clots which occur so frequently in the interior of the heart. Sometimes, on the other hand, it undergoes changes resembling those which fibrinous deposits in the interior of an aneurism undergo; it contracts and hardens, and blending with the arterial walls, forms ultimately with them a mere fibrous cord. Sometimes again, the obstructing clot becomes wholly or in part removed, and the channel of the vessel consequently restored.

It was formerly supposed that clots forming at the seat of inflammation, in arteries and veins, were due in great measure to inflammatory exudation taking place from the lining membrane of the affected vessel, and the experiments of Gendrin seemed to prove the validity of this explanation. But Virchow's more recent investigations, the results of which seem to have been confirmed by Henry Lee and Callender, show that if a vein, from the interior of which blood is excluded, be irritated, the substance of its walls becomes infiltrated with inflammatory products, but no exudation whatever takes place on the surface of the lining membrane. It is certain, therefore, that the lining membrane of veins, and, it may be added, that of arteries, either never furnish any inflammatory exudation, or furnish it rarely and with difficulty, and almost certain, therefore, that the plugs which are associated with arteritis are due to the mere coagulation of blood.

But even if it be admitted that these plugs are merely coagulated blood, it by no means follows that they are not in many cases the onsequence of arteritis. It is impossible, indeed, on any other view to explain how it is that in Arteritis commencing from without, clots form in the interior of the affected portion of vessel. Doubtless the lining membrane of the artery particles in the inflammatory process and the formation of a clot at the site of inflammation is due to some coagulating influence which the affected wall hence exerts over the blood with which it is in contact.

The causes of arteritis are as various as the causes of most other inflammations. Sometimes it is the consequence of irritation acting from within, as when an accidental thrombus forms at the part or an embolus becomes impacted there. More frequently it is the result of some cause acting locally, but from without, as when an artery is mechanically or otherwise injured, when it becomes surrounded by or imbedded in cancerous or tubercular formations, or when it become involved in suppuration of tissue, or in any other process of inflammation. Sometimes again, it is doubtless due to special conditions o general unhealthiness; and there is some reason to believe that certai forms of anæmia, and that syphilis, may be enumerated among them.

The results of arteritis are also various. Sometimes, no doubt, th arterial walls return to their original healthy condition; sometime they become indurated and perhaps thickened, with loss of contractil power and of elasticity; when obstructed by clot, they sometimes, a before stated, remain permanently obstructed, and if life be prolonge become reduced to mere fibrous cords; sometimes, on the other hand their walls become softened and ulcerated, and perforation may tak place, followed by hæmorrhage, and its consequences.

The most important results, however, of Arteritis are generall those secondary phenomena which take place, in consequence of th obstruction of the artery, in those tissues or parts to which th obstructed artery leads. These will be best considered under the hea of "Thrombosis and Embolia."

The symptoms, immediately due to Arteritis, may be very briefl enumerated. They consist in a greater or less amount of genera febrile disturbance, with (if the artery be within reach) pain an tenderness at the seat of inflammation, and perhaps fulness an hardness and loss of pulsation. But these rarely exist in an uncomplicated form; for on the one hand they are often, from the ver earliest moment, mixed up with, and masked by, the symptom of that morbid condition with which they are associated, or out which they have arisen; while on the other hand, when inflammatio affects an artery leading to any important part, the symptoms due t its obstruction soon by their gravity overshadow the symptoms du directly to arteritis.

The treatment of simple Arteritis may also be dismissed in a ver few words. It should consist, first, of such constitutional managemen as is appropriate for other forms of inflammatory affections, and secon (when the artery affected occupies a limb or is otherwise accessible) local abstractions of blood by leeches or otherwise, and according circumstances, of moist warmth, such as may be produced by poultice or of cold applications, especially of ice.

In speaking of Arteritis we have hitherto avoided all reference t those morbid conditions of the internal coat which Vircho regards as the results of endo-arteritis, and which are so frequently th seat of atheromatous and other degenerative changes. These consi of cartilage-like, slightly translucent circumscribed thickenings the internal membranes, which vary in size, are more or less rounde in outline, and project in a wheel-like form into the arterial cana and are found under the microscope to be due mainly to overgrowt of the cell-elements of the part. This latter fact shows that the are growths, and not, at all events in their earlier stages, degenerations and since they are developed slowly, and at the same time (at a events when they are limited to the larger arteries) without obvious symptoms, they may doubtless be regarded as chronic inflammatory changes, allied to those which constitute cirrhosis.

Again, laminæ of true bone are occasionally formed in the lining membranes of arteries; as also is that of veins. And here, as in the last case, we have obviously to deal, not with degeneration, but with growth and development. The laminæ are the result of some slow inflammatory process, or at all events of some process related to inflammation.

(2.) *Degeneration.*—Degenerative changes occur mainly in the internal and middle coats of arteries, and are for the most part attended with the deposition of fatty or of calcareous particles, and the gradual distinction of the tissues in which the particles accumulate. Degeneration is sometimes a primary change, but more frequently probably the result of antecedent chronic endo-arteritis. In the former case the normal structural elements of the arterial walls immediately decay, in the latter the decay takes place in the substance of a something due to the overgrowth of such elements. This distinction, which Virchow clearly demonstrates, is undoubtedly a real one; at the same time it is, as yet at all events, of little practical importance; for the two conditions are constantly associated, and they lead to the same issues.

Fatty degeneration of arteries usually goes by the name of atheroma. Virchow, who points out the inappropriateness of this term as usually employed of arterial disease, endeavours to limit its use to those cases in which the degeneration occurs in the deeper layers of the internal coat and leads to the formation there of a cavity containing fatty detritus—cases in which, as a matter of fact, the degeneration is almost without exception secondary. And he calls those cases fatty erosions in which the process begins on the free surface, and in which as a rule the degeneration is primary. But for reasons which have been already referred to we shall employ the word atheroma in its ordinary sense.

Atheromatous degenerations appear in the early stage as opaque white, or yellowish-white spots, seated either in the substance of the internal coat, or between that and the middle coat. They are irregular in size and shape, and form elevations, which encroach more or less on the arterial canal, but present, nevertheless, a perfectly smooth and polished free aspect. They are at this time probably few in number and widely scattered, but there are certain situations which they specially affect, among which are the margins of the orifices of origin of branches. At a later stage, they have increased in extent, partly by the formation of fresh spots, partly by the coalescence of neighbouring ones. Their thickness has at the same time increased and increased irregularly; so that the inner surface of the affected artery assumes an uneven and more or less tuberculated character. If a vertical section of the diseased arterial walls be now made, two or three varieties of condition will be observed, existing either alone, or more commonly in combination. In one case, the morbid process will be found to be entirely superficial. In another case the degen rations will be found to involve the deeper layers of the internal tuni The membrane at the seat of disease will then be found thickened two, three, or four times its original thickness, sometimes to a great degree still, attaining it may be in the aorta a thickness of a quart of an inch. It will be found opaque, yellowish-white, softer tha natural, pulpy or even semi-fluid in consistence; and it will, unle the softening be sufficient to destroy this characteristic, be found consist of superposed laminæ, arranged nearly parallel with the surfac and continuous at their edges with the laminæ of the healthy portic of the membrane. It will generally be observed, also, that a thi layer of comparatively healthy membrane separates the more disease tissue from the canal of the artery. In another case, but mo rarely, the morbid process attacks the middle coat, and may be limite to that coat.

The further changes which take place in atheromatous patch may be briefly stated. The more superficial ones soften on the surfac become eroded, and then gradually disappear, leaving an ulcer-lil pit or depression. The deeper-seated sometimes soften, and present discharge their contents through a small orifice in the lining men brane into the channel of the artery. Sometimes it would seem th more or less of the atheromatous material gets absorbed, and tl surface assumes in consequence an irregularly depressed, pucker appearance. Sometimes the diseased patches undergo cretification ar the artery assumes in their situation the so-called "ossified" conditio

Calcareous degeneration of arteries generally accompanies, or rath follows on, atheromatous deposit. Sometimes, however, it seems be developed independently of atheroma. Like atheroma, it general commences in the internal coat, and may be limited to it. It forn hard brittle plates of irregular shape, and of various sizes, whic in the first instance are limited for the most part to the deeper laye of the internal coat. At all events, in the early stage, the calcareo plates are separated from the current of blood by a thin layer of tl lining membrane. After a while, however, this layer becomes al infiltrated, and the earthy matter comes into direct contact with tl blood. The calcareous accumulations then form plates corresponding their general shape to the portion of the artery in which they ha been formed, having a tolerably smooth internal surface, and an out surface, which is more or less nodulated; occasionally they for nodulated projections even on the free surface. Not infrequent erosion of the artery takes place at the circumference of such plat and their rough edges become exposed to the blood; sometimes t plates become detached. In some of the smaller arteries more pa ticularly, as in those of the extremities, calcification takes place in t middle coat sometimes primarily, sometimes in association with simil disease of the internal coat. In this case the calcareous matter tends assume the form of rings, and obviously infiltrates the muscular fibr of the part.

Whenever atheromatous or earthy deposition takes place, even if it be confined to the lining membrane, some change in the nutrition of the middle tunic seems either to accompany it, or to follow quickly upon it. For the portion of this tunic which is subjacent to the deposit, even if it looks healthy, loses more or less completely both its elasticity and its contractile power. It need scarcely be added, that this loss of power occurs also, and even more markedly, when the middle tunic is distinctly involved in degeneration.

Atheromatous degeneration consists essentially in the transformation of the cellular elements of the tissue into oil (olein) and cholesterine, and the softening and breaking down of the intervening parts. The oil (which is said to be olein) occurs in the form of globules of various sizes, some so small as to constitute mere molecular matter, others so large as to exceed the size of a pus-corpuscle. The cholesterine occurs in two conditions; in one it forms the ordinary thin imperfect rhomboidal plates; in the other (mixed probably with albumen), it forms globules of various sizes, which, like those in the so-called fatty kidney, present a cross when examined with polarized light.

At some period in the course of atheromatous degeneration, the degenerated patches begin often to be the seat of calcareous depositions, they shrink probably in thickness and gradually concrete. This earthy transformation consists in the gradual deposition, among the other degenerative products, but chiefly in the intercellular tissues of earthy globules, and, at the same time, of the gradual disappearance by liquefaction, and absorption probably, of the cholesterine and oil. The earthy globules run together, blend into botryoidal masses, and at length form patches of earthy material which still give evidence of their mode of origin in the persistence of irregular angular cavities, in the nodulated character of their margins, and in the presence of free earthy globules in the neighbourhood of these margins. The process of calcification here described is similar to that observed in other forms of pathological calcification, as on the surface of serous membranes, in the teeth, and so on; it is similar also to the mode of calcification which Mr. Rainey has shown to be normal in shells, and in various other forms of healthy hard tissues, including bones. Pathological calcifications of this kind in many tissues, and even in veins, have been shown to become occasionally converted into true bone, so that true ossification of an artery is by no means an improbable event. Virchow, also looking at the question from a somewhat different point of view, considers that the earthy depositions taking place in the lining membranes of arteries result in true ossification. When calcareous deposit takes place independently of atheroma, or so much in excess of oily deposit as from the beginning to mask the presence of the latter, the process of calcification is still essentially the same as that which has been described. The earthy matter is deposited, that is to say, in globular particles and masses, which, by further deposition *on their* surface, grow together. When the calcareous change takes place in the middle coat the muscular fibres (as has before been pointed out) are the specia seat of this change and become converted into rigid spindle-shape bodies.

All arteries are liable to the various forms of atheromatous an earthy degeneration which have just been considered, but they are no all equally liable. Again, although, when the disease is presen there is a tendency for it to become general, it by no means ver infrequently happens that it is limited to one particular section the arterial system, and attains there even a very advanced conditio Among the arteries specially liable to be affected, and in which as rule the disease is found most developed, may be enumerated th aorta, the large trunks immediately springing from the aorta, th arteries of the brain, the coronary arteries of the heart, the splen artery, and the arteries of the extremities. To these may be adde disused arteries, such as those of stumps, and the arteries distribute in the walls of the senile uterus.

When atheromatous and calcareous deposits are at all extensive, th lead to many more or less important results. Thus, they produce oft extreme irregularity of surface, and sometimes to these irregulariti clots become adherent, and occasionally undergo that central softeni which will be found described in connection with cardiac clots ofte especially in small arteries (such as those of the brain, of the hea or of the extremities), the lining membrane becomes so much thic ened, that the passage of the blood becomes impeded, or even alt gether arrested; sometimes, the deposit of calcareous matter becomes extreme, that entire arteries are converted into rigid cylinders; som times again, the atheromatous degeneration is so abundant, that t softened lining membrane forms in the interior of the artery number of undulating partially detached excrescences, projecti loosely into its interior. And it is worthy of remark, that most the conditions just referred to occur not simply in arteries of lar or of medium size, but even in the minutest arteries and in t capillaries. The latter two kinds of vessel, it is well known, oft undergo the fatty form of degeneration; but they also occasiona undergo the purely calcareous form of degeneration. We have se them in the brain converted into rigid needle-like tubes, in whi under the microscope, the calcareous matter consisted entirely calcareous globules, which were deposited apparently beneath t lining membrane, and had coalesced more or less completely with o another.

The causes of these degenerative processes in arteries are obscu It is certain that they constantly attend on old age; and th according to the situation of the vessels chiefly affected, lead cerebral apoplexy, to senile gangrene of the extremities, to ang pectoris, or other forms of cardiac disease, or to aneurisms. it is also certain that they do not attend on old age exclusive that they are occasionally observed in infancy; and that they

[illegible]

[illegible]

[illegible]

There is another form [illegible] degeneration [illegible] as we know has only been [illegible] vessels and in the smallest arteries [illegible] of the vascular system [illegible] in the blood-vessels. It is seen [illegible] liver and in the spleen, when these viscera [illegible] form of degeneration is present [illegible] the capillaries and small vessels [illegible] other tissues of these organs. The affected vessels [illegible] and pellucid, and glassy-looking [illegible] waxiness, assuming a peculiar reddish-brown [illegible] tained some years since, that the matter deposited in waxy degeneration is cellulose; this view, however, has been [illegible] has since retracted it. Dr. Edmund M[illegible] several years ago, at the Pathological Society that [illegible] kid material contained much cholesterine [illegible] matter. And he regarded it as identical or nearly so [illegible] composition with the fatty-looking globules, acted on by polarized light, which occur amongst other globules in ordinary atheroma.[1]

Waxy degeneration is due apparently to the influence of certain conditions of system; especially of those which attend the later stages of syphilis, and chronic tubercular diseases, and of that which is induced by wasting suppuration, especially when bones are involved.

(3.) *Changes of Dimension.*—Alterations in the calibre of arteries take place very frequently, both as physiological and as morbid processes. As instances of the former may be mentioned, the enlargement of the collateral arteries of a limb in consequence of the obliteration of the main trunk, the alternate enlargement and diminution of the uterine arteries which attend the development and subsequent shrinking of the gravid uterus; and the atrophy which ensues after amputation in the arteries of stumps. The latter class of changes, however, the class (that is to say) which includes morbid dilatations and morbid contractions, is that which mainly interests us here.

(*a*) *Enlargement.*—An artery may be more or less generally enlarged, under which circumstance it is commonly spoken of as being dilated; or it may be enlarged at one or more distinct points, and the word aneurism is then employed to designate every such enlargement.

Dilatation.—Dilatation, in the sense just indicated, is not uncommonly met with in the degenerated arteries of old persons, especially perhaps in the aorta, and in the arteries at the base of the brain. The arteries thus affected are generally somewhat unevenly dilated, and present in consequence an irregularly nodulated or knobby contour. Dilatation is apt to occur also in a series of arteries belonging to some circumscribed locality, when by their aggregation they produce that condition which is commonly known by the name of cirsoid aneurism, or aneurism by anastomosis. The arteries in this disease, which are generally in their origin arteries of minute size, become generally and extremely dilated, they elongate and become tortuous, and ultimately by the formation of new anastomoses, or the enlargement of those which originally existed, communicate freely with one another in al directions. This dilatation does not appear to be attended with any structural disease of the arterial walls. They are, however, probably always very much thinner than those of healthy arteries of the same size.

Aneurism.—Circumscribed dilatations of arteries, or aneurisms, present many varieties of character dependent on their causes, the structure of their parietes, their form, and some other conditions. Hence in reference to their origin, they have been called idiopathic or traumatic, in reference to their walls, true or false or diffused, and in reference to their form, sacculated or fusiform; and they have been

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[illegible] words, and have [illegible] d as false, and that [illegible] ue and false are still [illegible] . Obviously it would be [illegible] m "diffused aneurism" [illegible] by some, it has been used to signify the diffusion of blood takes place when an artery [illegible] ruptures [illegible] the ar tissue—a condition to which as Mr [illegible] properly [illegible] rm of ruptured artery [illegible] is properly applicable [illegible] ver, it is employed to designate those cases in which after [illegible] pture the patient survives sufficiently long for the space [illegible] blood has escaped to become circumscribed by inflammatory ation, and thus to be converted into a cavity maintaining a free munication with the ruptured vessel. In this latter sense we shall tinue to employ it.

An ordinary or *Sacculated Aneurism* is a circumscribed dilatation of artery, involving generally a well-defined area of the arterial walls, d limited generally to one side, or a portion of one side only. When all in proportion to the artery from which it springs, it may be re or less hemispherical, or thimble-shaped, its fundus being its adest part. But sometimes while it is yet small and generally en it becomes large, it assumes a rounded or even arteries yield the largest aneurisms; but this is a rule liable to exceptions, of which, perhaps the most notable is, that aneurisms springing from the commencement of the aorta are almost always fatal from rupture while they are still very small. They vary, roughly speaking, from the size of a child's, or of an adult's, head to that of a pea or less. Their orifice is generally round or oval, though perhaps somewhat irregularly so. Sometimes this is bounded by a well-defined or tumid margin; but sometimes the cavity of the artery gradually dilates into the aneurism, and the orifice (though still defined above and below by an abrupt line) is undistinguishable laterally, and formed simply by the divergence in this situation of the arterial walls. Various descriptions have been given of the composition of the walls of such aneurisms as are under consideration. Some authors, indeed, enumerate every conceivable variety of composition, from that in which it is formed of all three coats, to that in which it is formed of one coat only. We need not follow them. As a rule, an idiopathic aneurism, when in an early stage of formation, or while it remains small, presents a lining membrane which is continuous at the margins of the aneurismal orifice with the lining membrane of the artery; and if the artery be macerated, or is slightly decomposed, the membrane may be detached, and will be found to form a complete cast of the cavity. This lining membrane is generally thicker and softer than that of the artery itself, and perhaps more translucent; but it presents little or no structural difference. In larger aneurisms the inner surface is still generally, in the greater part of its extent, more or less polished; but though still necessarily continuous with that of the artery, becomes for the most part quite inseparable from the tissues which it lines. Very often, even in commencing aneurisms, the middle coat of the artery terminates in the thickened rim which bounds its orifice; sometimes it may be traced for a variable and even considerable distance on to the aneurism; and sometimes flakes, of what appears to be the middle coat, are scattered here and there irregularly over the whole circumference of the tumour. It may be stated that, as a rule, the middle coat is either deficient or presents traces of its presence only. The external arterial tunic is that which mainly and most uniformly forms the wall of any aneurism. At first probably it exists there in an unaltered condition. But as the tumour grows, and as this coat becomes stretched, additional connective tissue becomes added to it and incorporated with it so that it becomes thicker and more resisting than the outer coat of the artery itself. And as the tumour continues to grow and presses upon neighbouring organs and tissues, these or portions of them become compressed and indurated, and contribute to the formation o its walls.

A *Diffused Aneurism* may be of any size or any shape; it may originate as such directly from an artery, and not very unfrequently it becomes superadded to an ordinary aneurism as a consequence o rupture of its walls; indeed, it is a very common thing to observe, in

[illegible]

[illegible]

[illegible]

Aneurisms may be produced by accident or spontaneously, and perhaps not very unfrequently in the course of [illegible] of an aneurism [illegible] In other words, accidental [illegible] are more likely to produce aneurisms in arteries which are already diseased and in a condition favourable to the spontaneous origin of aneurism, than they are in healthy arteries; and when aneurisms have already formed, their enlargement is not very unfrequently in part due to the occurrence of accidental ruptures. Accidental aneurisms are [illegible] in the popliteal artery, and other large arteries of the extremities, and in the aorta, as the result of a strain in which [illegible] that has been lacerated; and may occur in any artery as the consequence of a wound. In cases of idiopathic aneurism the essential cause of the disease is, doubtless, deficiency of resisting power in the middle coat of the artery at the seat of aneurism, as compared with the distending power of the blood to which it is opposed. The operation of this cause is shown in its simplest form in the instance already [illegible] in which an aortic arch, otherwise healthy, becomes dilated in consequence of the obliteration of its further extremity. [illegible] dilatation and idiopathic aneurism most commonly [illegible] in connection with atheromatous and osseous deposits. What [illegible] together. When the calcareous change takes place in the middle coat, the muscular fibres (as has before been pointed out) are the special seat of this change and become converted into rigid spindle-shaped bodies.

All arteries are liable to the various forms of atheromatous and earthy degeneration which have just been considered, but they are not all equally liable. Again, although, when the disease is present there is a tendency for it to become general, it by no means very infrequently happens that it is limited to one particular section of the arterial system, and attains there even a very advanced condition. Among the arteries specially liable to be affected, and in which as a rule the disease is found most developed, may be enumerated the aorta, the large trunks immediately springing from the aorta, the arteries of the brain, the coronary arteries of the heart, the splenic artery, and the arteries of the extremities. To these may be added disused arteries, such as those of stumps, and the arteries distributed in the walls of the senile uterus.

When atheromatous and calcareous deposits are at all extensive, they lead to many more or less important results. Thus, they produce often extreme irregularity of surface, and sometimes to these irregularities clots become adherent, and occasionally undergo that central softening which will be found described in connection with cardiac clots often especially in small arteries (such as those of the brain, of the heart or of the extremities), the lining membrane becomes so much thickened, that the passage of the blood becomes impeded, or even altogether arrested; sometimes, the deposit of calcareous matter becomes s extreme, that entire arteries are converted into rigid cylinders; sometimes again, the atheromatous degeneration is so abundant, that th softened lining membrane forms in the interior of the artery a number of undulating partially detached excrescences, projectin loosely into its interior. And it is worthy of remark, that most o the conditions just referred to occur not simply in arteries of larg or of medium size, but even in the minutest arteries and in th capillaries. The latter two kinds of vessel, it is well known, ofte undergo the fatty form of degeneration; but they also occasionall undergo the purely calcareous form of degeneration. We have see them in the brain converted into rigid needle-like tubes, in whic under the microscope, the calcareous matter consisted entirely o calcareous globules, which were deposited apparently beneath th lining membrane, and had coalesced more or less completely with on another.

The causes of these degenerative processes in arteries are obscur It is certain that they constantly attend on old age; and the according to the situation of the vessels chiefly affected, lead t cerebral apoplexy, to senile gangrene of the extremities, to angin pectoris, or other forms of cardiac disease, or to aneurisms. Bu it is also certain that they do not attend on old age exclusivel that they are occasionally observed in infancy; and that they n

ictly marked. Persons whose arteries are in this condition
ten for years while the disease is in progress, and yet suffer
nconvenience from it. The symptoms, indeed, which call
on to the presence of degeneration are almost entirely due to
nditions which complicate degeneration, to rupture, to aneurism,
truction of arteries, to enlargement of the heart, and so on.
ometimes, when the superficial arteries are the seat of degenera-
he fact that they are so is indicated by their form and their
y; sometimes, when the cerebral arteries are affected, transient
symptoms point to the presence of this condition; sometimes,
the aorta is the special seat of mischief, something in the
n, in the sounds, and in the dimensions of the heart, suggests
esence of aortic disease.

re is even less to be said in regard to the treatment of arterial
ration, than in regard to its symptoms. All that need be
on this head is, that the patient in whom such arterial
e is suspected should guard against all excitement, mental
odily, that he should give way to no excess of any kind,
hat he should endeavour to live quietly, and regularly, and
rately.

re is another form of arterial degeneration, known as the waxy,
eous, or so-called "amyloid" degeneration, which, so far
know, has only been hitherto detected certainly in the capillary
s, and in the minutest arteries. So far as this is a disease
vascular system, it may be regarded in the light of a patho-
l curiosity only, inasmuch as it leads to no symptoms referable
blood-vessels. It is seen constantly in the kidneys, in the
and in the spleen, when these viscera are the subjects of the
of degeneration in question. It seems, indeed, to commence in
pillaries and small vessels, and to involve subsequently only the
tissues of these organs. The affected vessels become thickened

composition with the fatty-looking globules, acted on by polarized light, which occur amongst other globules in ordinary atheroma.[1]

Waxy degeneration is due apparently to the influence of certain conditions of system; especially of those which attend the later stages of syphilis, and chronic tubercular diseases, and of that which is induced by wasting suppuration, especially when bones are involved.

(3.) *Changes of Dimension.*—Alterations in the calibre of arteries take place very frequently, both as physiological and as morbid processes. As instances of the former may be mentioned, the enlargement of the collateral arteries of a limb in consequence of the obliteration of the main trunk, the alternate enlargement and diminution of the uterine arteries which attend the development and subsequent shrinking of the gravid uterus; and the atrophy which ensues after amputation in the arteries of stumps. The latter class of changes, however, the class (that is to say) which includes morbid dilatations and morbid contractions, is that which mainly interests us here.

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Dilatation.—Dilatation, in the sense just indicated, is not uncommonly met with in the degenerated arteries of old persons, especially perhaps in the aorta, and in the arteries at the base of the brain. The arteries thus affected are generally somewhat unevenly dilated, and present in consequence an irregularly nodulated or knobby contour. Dilatation is apt to occur also in a series of arteries belonging to some circumscribed locality, when by their aggregation they produce that condition which is commonly known by the name of cirsoid aneurism, or aneurism by anastomosis. The arteries in this disease, which are generally in their origin arteries of minute size, become generally and extremely dilated, they elongate and become tortuous, and ultimately by the formation of new anastomoses, or the enlargement of those which originally existed, communicate freely with one another in all directions. This dilatation does not appear to be attended with any structural disease of the arterial walls. They are, however, probably always very much thinner than those of healthy arteries of the same size.

Aneurism.—Circumscribed dilatations of arteries, or aneurisms, present many varieties of character dependent on their causes, the structure of their parietes, their form, and some other conditions. Hence in reference to their origin, they have been called idiopathic or traumatic, in reference to their walls, true or false or diffused, and in reference to their form, sacculated or fusiform; and they have been classified by different authors in accordance with one or other of the plans thus indicated. The several names just enumerated are in common use. Of them, some are employed only in their literal sense, and scarcely need, therefore, any explanation; but two or three of them not only convey no very obvious meaning, but have been used in such different, nay opposite, senses by different authors, that their retention is a source of constant confusion. The names to which we here specially refer are those of true, false, and diffused aneurisms. By Scarpa, and other earlier writers, the term true was applied to those ordinary forms of aneurism in which, as a rule, the middle coat of the artery is deficient, or in which, at all events, the walls of the aneurism do not include all the layers constituting the arterial wall; and the term false was used of that comparatively rare form of aneurism, in which the walls are formed of all the arterial tunics. Hodgson, however, and subsequent writers on the subject, have, unfortunately, exactly transposed the use of these words, and have called that true aneurism which Scarpa described as false, and that false which he described as true. The terms true and false are still constantly employed, and generally inexactly. Obviously it would be convenient to drop them altogether. The term "diffused aneurism," again, has been used in various senses. Thus, by some, it has been used to signify that diffusion of blood which takes place when an artery, or an aneurism, ruptures into the cellular tissue—a condition to which, as Mr. Holmes properly insists, the term of ruptured artery or aneurism is properly applicable. By others, however, it is employed to designate those cases in which after such a rupture the patient survives sufficiently long for the space into which the blood has escaped to become circumscribed by inflammatory induration, and thus to be converted into a cavity maintaining a free communication with the ruptured vessel. In this latter sense we shall continue to employ it.

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An ordinary or *Sacculated Aneurism* is a circumscribed dilatation of an artery, involving generally a well-defined area of the arterial walls, and limited generally to one side, or a portion of one side only. When small in proportion to the artery from which it springs, it may be more or less hemispherical, or thimble-shaped, its orifice being its broadest part. But sometimes while it is yet small, and generally when it becomes large, it assumes a rounded, or even completely globular form, and then opens into the artery with which it is connected by an oval orifice, less in breadth than the aneurism itself—sometimes very much less—and with its long diameter corresponding in direction with the channel of the artery. Sometimes from the enlarging sac yielding more readily at one or more points than elsewhere, the original aneurism may have other aneurisms, as it were, springing from it; sometimes the irregular resistance opposed to its growth by surrounding parts compels its enlargement in certain directions rather than in others; and from these, or other causes, aneurisms may come to assume almost any variety of shape. Their size varies within very wide limits. As a rule, it may be considered that the largest arteries yield the largest aneurisms; but this is a rule liable to exceptions, of which, perhaps the most notable is, that aneurisms springing from the commencement of the aorta are almost always fatal from rupture while they are still very small. They vary, roughly speaking, from the size of a child's, or of an adult's, head to that of a pea or less. Their orifice is generally round or oval, though perhaps somewhat irregularly so. Sometimes this is bounded by a well-defined or tumid margin; but sometimes the cavity of the artery gradually dilates into the aneurism, and the orifice (though still defined above and below by an abrupt line) is undistinguishable laterally, and formed simply by the divergence in this situation of the arterial walls. Various descriptions have been given of the composition of the walls of such aneurisms as are under consideration. Some authors, indeed, enumerate every conceivable variety of composition, from that in which it is formed of all three coats, to that in which it is formed of one coat only. We need not follow them. As a rule, an idiopathic aneurism, when in an early stage of formation, or while it remains small, presents a lining membrane which is continuous at the margins of the aneurismal orifice with the lining membrane of the artery; and if the artery be macerated, or is slightly decomposed, the membrane may be detached, and will be found to form a complete cast of the cavity. This lining membrane is generally thicker and softer than that of the artery itself, and perhaps more translucent; but it presents little or no structural difference. In larger aneurisms the inner surface is still generally, in the greater part of its extent, more or less polished; but though still necessarily continuous with that of the artery, becomes for the most part quite inseparable from the tissues which it lines. Very often, even in commencing aneurisms, the middle coat of the artery terminates in the thickened rim which bounds its orifice; sometimes it may be traced for a variable and even considerable distance on to the aneurism; and sometimes flakes, of what appears to be the middle coat, are scattered here and there irregularly over the whole circumference of the tumour. It may be stated that, as a rule, the middle coat is either deficient or presents traces of its presence only. The external arterial tunic is that which mainly and most uniformly forms the wall of any aneurism. At first probably it exists there in an unaltered condition. But as the tumour grows, and as this coat becomes stretched, additional connective tissue becomes added to it and incorporated with it so that it becomes thicker and more resisting than the outer coa of the artery itself. And as the tumour continues to grow and presses upon neighbouring organs and tissues, these or portions of them become compressed and indurated, and contribute to the formation o its walls.

A *Diffused Aneurism* may be of any size or any shape; it may originate as such directly from an artery, and not very unfrequently i becomes superadded to an ordinary aneurism as a consequence o rupture of its walls; indeed, it is a very common thing to observe, in large aneurisms, that the proper parietes are here and there, over some well-defined area, deficient, and replaced by condensed, blood-infiltrated tissue belonging to the parts in which the aneurism is imbedded. The parietes of a diffused aneurism consist simply of the tissues which happen to have limited the escape of blood, and which have become to some extent infiltrated with blood, indurated and, according to their age, more or less fibrous.

A *Fusiform Aneurism* is an aneurism in which the entire circumference, or the greater part of the circumference, of an artery becomes for a limited and tolerably well-defined part of its length, dilated. Such an aneurism is generally more or less irregular in form, and indeed approximates in structure and in mode of formation to that condition which we have already described under the head of Dilatation. It may, however, like an ordinary sacculated aneurism, become very large; and this enlargement may be due either to its progressive general increase, or to the superaddition of a sacculated aneurism. As might be supposed, aneurisms of this kind may comprise in their parietes all the arterial coats, and probably in the beginning always do so; but, in their onward progress, their parietes naturally tend to become identical with those of the sacculated variety.

A modification of the fusiform aneurism is occasionally observed in the aortic arch in cases where the aorta is greatly contracted or obliterated at the point of entrance of the ductus arteriosus. The arch in these cases becomes generally and considerably dilated, sometimes so much so as to be capable of containing the fist; the walls undergo great attenuation, but (excepting accidentally) remain free from atheromatous or other unhealthy deposit, and whole.

Aneurisms may be produced by accident, or spontaneously; and perhaps not very unfrequently in the course of the complete development of an aneurism both causes may have operated in various degrees. In other words, accidental occurrences are more liable to produce aneurisms in arteries which are already diseased, and in a condition favourable to the spontaneous origin of aneurism, than they are in healthy arteries; and when aneurisms have already formed, their enlargement is not very unfrequently in part due to the occurrence of accidental ruptures. Accidental aneurisms are often met with in the popliteal artery, and other large arteries of the extremities, and in the aorta, as the result of a strain, in which probably the middle coat has been lacerated; and may occur in any artery as the consequence of a wound. In cases of idiopathic aneurism, the essential cause of the disease is, doubtless, deficiency of resisting power in the middle coat of the artery at the seat of aneurism, as compared with the dilating power of the blood to which it is opposed. The operation of this cause is shown in its simplest form in the instance already quoted, in which an aortic arch, otherwise healthy, becomes dilated in consequence of the obliteration of its further extremity. But idiopathic dilatation and idiopathic aneurism most commonly arise in connection with atheromatous and ossific deposits. When these are all extensive, the middle coat, even when not obviously itself the seat of such deposits, loses both its contractile and elastic powers, and is then apt to yield under the constant impulse of the blood. It becomes attenuated, its fibres separate from one another, and, as the tumour becomes larger, either wholly or in part disappear. For a time, no doubt, the thickening of the inner coat, due to atheromatous or earthy change in it, protects the feeble middle coat from the injurious operation of the dilating force. But, after a while, the atheromatous inner coat becomes eroded and removed, or the atheromatous collection between it and the middle coat becomes discharged into the artery, or the bony plate becomes partially or entirely detached; and in one of these ways, or in some other way, the protecting influence is removed from the enfeebled middle coat, which then begins to expand. It is not, however, absolutely necessary that the removal of the internal coat should even in cases of atheroma precede the formation of aneurism; it is merely necessary that its own resisting power should not be sufficiently great to compensate for the loss of this power in the middle coat. It may be added, that any other condition tending to enfeeble the middle arterial tunic may be a cause of aneurism. Thus aneurisms arise as a result of inflammation involving the walls of arteries; and Tufnell, Holmes, and J. W. Ogle have all published cases in which aneurisms seem to have supervened in this way upon embolia. Sometimes an artery may be opened by ulceration commencing from without, and thus a diffused aneurism may be produced.

When once an aneurism has begun it almost always undergoes gradual enlargement. Up to a certain point, as has been already shown, its parietes are for the most part derived solely from those of the artery out of which it has originated; but soon, in its progress, it begins to press on and displace neighbouring organs, and then to appropriate them, as it were, in the formation of its walls. Its enlargement may be almost unlimited when it is developed in the substance of cellular tissue, as behind the peritoneum, or when its chief growth is subcutaneous; but when it projects towards serous surfaces, or presses upon mucous channels, it tends comparatively early to open into them. The ultimate tendency, indeed, of all aneurisms is to rupture. Often, as has been pointed out, partial ruptures occur into cellular tissue, and the additional cavities thus formed become circumscribed and taken into that of the original tumour. Often an aneurism opens by a sudden tear into a serous cavity, as that of the pericardium, the pleuræ, or the peritoneum; but perhaps more frequently the rupture first takes place into the sub-serous tissue, the blood accumulating therein and finally escaping thence by a subsequent rupture into the serous cavity itself. Often again an aneurism, after gradually pressing upon a mucous canal, opens into it through the formation and separation of a slough between them. In this way thoracic aneurisms frequently open into the trachea, bronchial tubes, or œsophagus; and abdominal aneurisms occasionally discharge themselves into some lower part of the alimentary canal. Occasionally, too (but this is usually a very late event), an aneurism bursts externally, having previously by its pressure caused the formation of a cutaneous slough. In rare cases an aneurism opens into an artery, a vein, or even into the heart itself. These latter events are most common in aneurisms of the aortic arch. But aneurisms produce mischief not merely by bursting and causing fatal hæmorrhage. They are apt, by pressing on various organs, to produce effects referable to these organs; thus when developed on the under surface of the brain, they may produce eclampsia, or interfere with vision or with hearing, or lead to some other nervous phenomena; thus when developed in the thorax they may compress the trachea or the œsophagus and so impede respiration or swallowing, they may involve the recurrent laryngeal nerve or the sympathetic, and induce certain characteristic symptoms which will be elsewhere detailed; and they may compress, and lead to the obliteration of venous trunks, and thus induce dilatation of tributary and anastomotic veins, and œdema of the parts beyond the seat of obstruction. The effect of aneurisms on bones is curious. It is well known that, as they enlarge and press upon bones, they cause their erosion and gradual disintegration; that in this way a thoracic aneurism will destroy more or less completely the bodies of two or three vertebræ, and may even, in consequence of this destruction, burst into the vertebral canal; that it will destroy portions of the sternum, of the ribs or of the clavicles. Whilst this destruction is in progress, the eroded surface of bone lies exposed in the aneurism, forming a segment of its walls; and not unfrequently a partially destroyed clavicle or rib is found projecting into the interior of an aneurism.

Occasionally aneurisms undergo spontaneous cure. To understand this it is necessary to consider the changes which take place within their cavities. Sometimes after death aneurisms are found empty, or filled only with imperfectly coagulated blood or mere *post-mortem* coagulum. Sometimes, on the other hand, they are lined with laminated clots. These consist of a series of more or less imperfect layers, which lie one within the other, concentric with the aneurismal walls. They are pretty firmly attached to one another, but admit of separation, and the outer one is generally somewhat firmly united with the lining membrane of the aneurism. They consist of a toughish fibrinous material, of which the outer layers are thin and more or less buff-coloured, the inner become thicker and softer and redder as they approach the channel through which the blood is still passing. The amount of these clots varies very much; sometimes there are merely two or three layers, so that the cavity of the aneurism is scarcely encroached on by them; at other times they fill the greater part of the cavity; and occasionally they are so abundant as completely to obliterate it: the innermost laminæ filling up the orifice of the aneurism and lying flush with the general surface of the artery, or even forming an irregular projection into its channel. Clots of this kind are very commonly observed in sacculated aneurisms, but are not invariably present in them, and very large sacculated a risms are sometimes wholly free from them. Fusiform aneurism almost always without them. It need scarcely be said that these are distinctly deposited from the blood. The cause of their depos is probably twofold. In the first place it is a recognised fact blood tends to coagulate upon rough or diseased surfaces; thus mere atheromatous patches occurring in an otherwise healthy ar coagula are sometimes seen to have formed. In the second pl from the fact that these coagula are far more common in aneuri which communicate with an artery by a comparatively small ori than in those which are mere fusiform dilatations, it may be ta for granted that stagnation of blood tends to encourage the deposit of fibrine. The formation of these clots depends then, probably, in so cases on one of these causes, in some on the other of these causes, generally, doubtless, upon the concurrence of the two. It may added that the presence of a layer of fibrine is probably the m efficient cause of all in determining fibrinous deposition; just as nucleus of calcareous matter in the bladder attracts to itself simi matter which otherwise would have been retained in solution in t urine.

The formation of these clots in quantity no doubt frequently oppos an important barrier to the growth of an aneurism; and as has bee shown they sometimes form so abundantly as completely to oblitera its cavity. In the latter way a spontaneous cure may sometimes l effected; but it is doubtful if such cures would generally prove pe manent; for they are mostly observed in persons who have bee confined to bed some time previous to death, and the clots on whic they depend are generally not very difficult of detachment. But indee on the other hand, it is occasionally in consequence of such a detach ment that an aneurism becomes cured; the mass of clot, or a portio of it, shifts its position, and blocks up the artery upon which th aneurism is seated, leading at the same time to obliteration of th arterial canal and of the aneurism. Sometimes, again, an aneurism becomes cured by itself compressing the arterial tube either above or below its orifice.

Aneurisms may occur in any artery; but much more frequently in certain arteries than in others. Generally, it may be said that those arteries which are most prone to atheromatous and earthy degenerations are those which are also most prone to aneurismal dilatation. Such are the aorta, especially its arch, the innominate, carotid, and subclavian arteries, the cœliac axis, the common iliacs, and the arteries at the base of the brain. But the proneness to aneurism is not solely in proportion to the proneness to degeneration; for in consequence of the comparative violence of the impulse to which arteries near the centre of circulation are exposed, these incur a special risk which those further removed escape; and again, in consequence of the position and connections of certain arteries (as the popliteal) they are exposed to danger of various kinds of violence to which many other arteries are exposed little if at all. But we repeat that aneurisms may arise in almost any artery. Besides those named, or indicated, aneurisms are met with in the mesenteric artery and its branches, in the coronary arteries of the heart, in the ophthalmic artery, and in one case we found one in the course of one of the small arteries in the substance of the cerebellum. The pulmonary system of arteries is very rarely the seat of aneurisms; still, they are occasionally met with both in its trunk and in its branches. These arteries are more frequently dilated.

Aneurisms occur much more frequently during middle age and in declining years than in the earlier periods of life; but they are far from uncommon (in the labouring classes especially) between the ages of thirty and forty. And still younger persons, even children, do not wholly escape. They affect men more frequently than women. This is especially true of aneurisms of the extremities, which are mostly the result of violence.

The local indications of an aneurism, when it is seated in some accessible part, consist in the presence of a dilating pulsatile tumour, attended frequently when it is developed in the course of large arteries by a murmur synchronous with the systole of the heart. But in the case of many aneurisms, as of those within the skull, and in other inaccessible situations, these indications fail us entirely. The other symptoms of aneurism can scarcely be usefully considered here, inasmuch as they are in part those due to degenerated arteries generally, in part those which are due to the pressure of the aneurism on surrounding organs and tissues (and these symptoms will necessarily vary with the situation of the aneurism), and in part also those which may arise from their rupture (and these again must vary according to the part into, or in connection with which, the rupture takes place).

It is difficult also to consider usefully in a short space the treatment of aneurism. Indeed, we are compelled to pass over in silence the important subject of their surgical treatment. With regard to aneurisms which are beyond the reach of surgery, the essential objects to be held in view are, first, to prevent their increase; and second, to promote their obliteration by the deposition of clots within them. These ends can only be attained, so far as we know, by maintaining as much as possible rest of body and of mind, especially by quieting the circulation and preventing any such bodily movements as are likely to affect the aneurism immediately. When aneurisms approach the surface, the application of ice or other sedatives over them, galvano-puncture, and the introduction even of foreign bodies, as of threads or wire, may aid coagulation. At one time it was supposed that the frequent abstraction of blood, and the use of a low diet, were important adjuncts in promoting the cure or retarding the progress of aneurisms. Such was Valsalva's method. But modern physicians, and among these must be specially mentioned Dr. Stokes, have generally found reason to disapprove of this plan of treatment, and to prefer (as it seems to us with reason) the use of a nutritious and even of a generous diet, abstinence from bleeding and other hurtful drains on the system.

Before concluding the subject of aneurism, it is necessary to c sider two or three abnormal conditions of artery to which the te Aneurism is with more or less inappropriateness commonly appli These are dissecting aneurism, aneurismal varix, varicose aneuris and cirsoid aneurism, or aneurism by anastomosis.

Dissecting Aneurism occurs chiefly, if not entirely, in the aorta. commences (generally in connection with degenerative disease, b sometimes when the vessel is simply dilated) in a rupture of t internal coat, through which blood is forced between the middle a outer coats, or rather (as Dr. Peacock has shown) into the substan of the middle coat, where it accumulates, separating the internal an external coats from one another. Sometimes the resulting separatio is slight, extending little beyond the ruptured orifice; sometimes it i very extensive, "dissecting" the coats of the aorta from the arch t the bifurcation, and extending even into the iliac arteries. Such a aneurism may after a while undergo a further rupture, either externally through the outer coat, or internally through the inner coat, and thus communicate by a second orifice with the interior of the artery. Sometimes the accumulation of blood in the substance of the walls is so great that the artery becomes obstructed.

Varicose Aneurism and *Aneurismal Varix*, are names which have been applied to the very rare conditions which follow on the establishment of a communication between a neighbouring artery and vein. Such a communication may (as the result of disease) take place between an aortic aneurism and the superior cava; and (as the result of accident) between certain of the arteries and veins of the extremities. It used occasionally to result from wounding the artery through the vein, at the bend of the elbow, in the operation of phlebotomy. To the case in which the artery opens immediately into the vein, the term aneurismal varix is applied; to that in which an aneurismal cavity lies between the communicating vessels, the term varicose aneurism.

Of *Cirsoid Aneurism* we have already spoken. This is sometimes a congenital affection; sometimes arises without any obvious cause at various times after birth, and even in adult life; and it may appear in various parts of the body, as in the orbit, in the fingers, and elsewhere, but most frequently in the scalp. A remarkable case is recorded in the "Pathological Transactions," where nearly the whole of one of the lower extremities was involved.

(*b*) *Contraction and Occlusion.*—These conditions may be produced in a variety of ways, of which some have already been partially considered. Their more important causes we will here enumerate, leaving what little we have to say about the symptoms and treatment of obstruction till we come to consider the subjects of thrombosis and embolia. Occlusion of arteries is sometimes a congenital defect; sometimes it is the result of injury; sometimes it arises from the pressure of some hard tumour growing external to the artery, or from the artery becoming compressed by, or involved in, some carcinomatous or other growth; not unfrequently it takes place in the course of atheromatous and earthy degeneration, as a consequence either of excessive thickening of the lining membrane of the artery, or of the partial detachment of diseased patches, or of the formation of clots in connection therewith. To such obstructions of the arteries of the lower extremities, senile gangrene is perhaps always due; a similar condition of the coronary arteries of the heart leads now and then to local patches of degeneration and to rupture of the heart; and a similar condition of arteries of the brain, to circumscribed softening of that organ. Lastly, occlusion is not unfrequently due either to the formation of clots in arteries (thrombosis) at the seat of obstruction, or to the impaction in arteries of clots and other matters brought to them from some comparatively remote portion of the vascular system (embolia). These last two causes of obstruction will be considered under the head of "Thrombosis and Embolia."

DISEASES OF VEINS.

By John Syer Bristowe, M.D., F.R.C.P.

(1.) *Inflammation.*—The term "Phlebitis" was formerly used, lik the term "Arteritis," in a much wider and much looser sense, an much more inaccurately, than it is for the most part at the presen day. It signified then a disease of the gravest import; for it was th common designation of most cases of what is now called pyæmia, o many obscure fatal cases in which it was erroneously supposed tha inflammation had crept from some distant vein to the heart, and o many cases of what would now be termed thrombosis or embolia. I included also true Phlebitis, or inflammation of the veins: the diseas which alone we now propose to consider.

Phlebitis, in this latter limited sense, is a morbid condition o vein, due sometimes to local, sometimes to constitutional, causes sometimes commencing from within the vein, sometimes from without attended with important changes in the venous walls, and frequentl with more or less of inflammatory mischief external to the vessel, an with coagulation of blood or other morbid phenomena within it.

The changes which indicate the presence of inflammation affec the outer vascular region of the venous walls in a far higher degre than they do the inner region which is devoid of capillary vessels When inflamed, the walls become thickened (sometimes several time thicker than in health), congested externally, infiltrated with inflammatory exudation, and softened; sometimes suppuration takes place i them, sometimes they become disintegrated, or ulcerated and pe forated, or entirely destroyed. If the inflammation be a chroni process, the exudation in the walls assumes a fibroid character, and th walls get indurated as well as thickened.

Generally while these changes are in progress, the connective tissu with which the vessel is surrounded, partakes in the inflammator processes, and consequently becomes congested, infiltrated, and, according to circumstances, indurated and brawny, or the seat of suppuratio At the same time, too, changes for the most part take place withi the vessel. Blood coagulates there and clings to its lining membrane a clot filling up, and it may be distending and occluding the vei mes thus established, and secondary phenomena due to the arrest ie circulation ensue. Clots thus formed undergo various changes, h will be afterwards more fully considered; sometimes they in solid, and gradually contract and indurate; sometimes they n and become reduced to a puriform fluid; and sometimes, we ve, they undergo actual suppuration.

ie causes of phlebitis are very various, and influence to a great it its degree, its character, and its progress. Thus, in some nces, it is due simply to the affected vessel being pressed upon, or unded, by some tubercular, cancerous, aneurismal, or other growth. generally the walls of the vein become greatly thickened and rated, and its channel filled with a clot, which is usually hard fibrinous, and closely adherent to the lining membrane. Some-, however, the vessel becomes flattened and so obstructed, and are formed only beyond the seat of obstruction. Sometimes, he portion of vessel chiefly involved becomes entirely destroyed no longer traceable.

many cases phlebitis is consequent on the vein being involved flammation (erysipelatous or other) affecting primarily the organ ssue in which the vein is included; and the phlebitis then tends rtake more or less in the character of the surrounding inflamma- The sheath of the vein becomes first inflamed, and subsequently nflammatory process invades successively the outer, middle, and nal coats; leading sometimes only to their congestion and to thickening, but sometimes resulting in distinct suppuration. ie latter case the sheath of the vessel may become uniformly infil- d with pus, or present more or less isolated collections of that , and pus may be developed in the substance of the outer tunics; occasionally as a result of this the venous walls undergo erosion lceration from without inwards, and a communication, or commu- ions, become established between the interior of the vein and arts external to it. When the inflammation extends thus from out, it sometimes happens that the outer walls only of the vein are ved; the walls become thickened in the aggregate, but the lining brane remains smooth and polished, no coagulum forms, and the nel remains free. More frequently, however, the walls become ted in their whole thickness, and at the seat of the disease a forms, which adheres and blocks up the canal of the vein. This as before stated, tends to soften either generally or in certain spots, thus to assume the character of an abscess, or of a collection of ounded on all sides by coagulum, or by a layer of inflammatory h. There is no doubt that in nearly all these cases the pus-like , and the more solid material bounding it, are simply the conse- ce of degenerative processes taking place in clots deposited from lood. But occasionally true pus is certainly met with in this tion. Sometimes this is due to the opening of an abscess into a and the consequent conversion of a limited portion of the venous m into an abscess; but sometimes it is due, we believe, either to the development of pus from the lining membrane of the vein, or to suppuration occurring in the substance of the clot. This latter condition we have undoubtedly observed in cases of erysipelas.

Again, phlebitis may result from local irritation, from poisoned wounds, or from other injuries. The inflammation then affects principally the sheath of the vessel, which becomes congested and infiltrated with inflammatory products, and sometimes undergoes suppuration; and it tends to travel along the sheath, so that presently a considerable length of vein, or a system of veins, may become affected. The same changes take place in this case in the venous walls, and in the interior of veins, as have been already considered.

In other cases again, the inflammation doubtless commences from within, the outer portions of the walls of the veins becoming involved secondarily to the inner. This is the case, probably, when without any cause, originating, so far as we can see, in the walls themselves, thrombi form in certain veins, as in the iliac veins, for example, of phthisical patients. Such thrombi distend and occlude the vessels which subsequently only to the formation of these thrombi become thickened and give other evidence of inflammation.

There are yet other cases, probably, in which thrombi form, wherein the inflammation associated with their formation is a primary inflammation of the venous walls, dependent probably on some constitutional affection. Such cases, however, are necessarily obscure, and it is difficult to distinguish them from those which have just been considered.

We may add here a few words on the different conditions of system in which phlebitis is most apt to supervene, and on the veins which are most prone to be affected. Phlebitis frequently takes place, as has been already stated, in the soft tissues, in connection with various forms of inflammation, such as erysipelas, diffuse cellular inflammation and carbuncle. It takes place frequently, too, in connection with inflammation, and especially with suppuration, of bones and joints; under this head may be included phlebitis of the lateral sinuses of the skull, consecutive to suppuration in the ear. It is peculiarly apt to supervene in the puerperal state, frequently affecting the uterine veins, and frequently also affecting other of the systemic veins, but more particularly the iliac, producing, or aiding to produce, the condition known as phlegmasia dolens. Phlebitis, too, is very liable to occur in the course of phthisis and of heart disease. In the former of these affections especially, it is not uncommon to meet with it in the iliac veins, and in the venous trunks connected with the upper extremities. It is also discovered now and then, both in these diseases and in others, in the renal veins, in some of the veins connected with the liver, and other visceral veins. Lastly, we may point out that phlebitis is liable to arise in veins already otherwise diseased, as, for example, in varicose veins.

Just as inflamed arteries, which have become obstructed with clot, lead in consequence of this obstruction to secondary affections in organs and tissues to which they are distributed, so inflamed veins, when they have become similarly occluded, produce in consequence of their occlusion secondary phenomena in the regions which they drain. The obstruction of a vein necessarily leads in a greater or less degree to stagnation of blood, first in the veins, and next in the capillaries which form part of its system. This stagnation of blood, with the consequent dilatation of the vessels in which it is stagnant, may be the only secondary phenomenon; and it may soon disappear, provided either the vein becomes pervious again, or the anastomotic veins are sufficiently large or numerous to take on readily its suspended functions. In other cases the veins beyond the seat of obstruction become dilated and tortuous, and serum exudes into the connective tissue, causing anasarca. This dilatation of tributary veins, with localized anasarca, is well seen, in the legs when the obstruction takes place in the iliac veins, in the arms when it occurs in the subclavian and axillary veins, and in the head and neck and arms when the venæ innominatæ are the seat of disease. But the formation of phlebitic clots is attended with a class of dangers different from those which have just been considered, and different from any which attend the formation of clots in arteritis. I allude to the dangers of embolia, to the dangers, that is to say, of the detachment of the phlebitic clots or of fragments of them, of their transference by means of the circulation to other parts of the system, of their impaction in small arteries, and of the effects more or less serious to which they may then lead, in the areas which the obstructed arteries happen to supply; and I allude also to the supervention of pyæmia which mostly, as before pointed out, has its starting-point in some local phlebitis taking place in connection with some unhealthy inflammatory process.

The local symptoms of phlebitis consist in pain and tenderness in the course of the affected portion of vein, with distinguishable fulness and hardness, if it be superficial, and often with redness or livid discoloration in the integuments over it. These symptoms are attended also with more or less general febrile disturbance, and followed soon, generally, by distension of the veins beyond, and by anasarca. If the phlebitis be of that kind which, commencing in the venous sheaths, tends to spread along them from the smaller to the larger veins, the symptoms become altogether more grave; the pain and tenderness, the hardness and superficial congestion, which mark the seat of disease, are observed to spread and to become extensive; abscesses may form here and there around the veins, and may even lay open portions of them; the constitutional symptoms are those of high fever, which tends to assume the typhoid character.

The symptoms of phlebitis, however, in complicated cases, are very often difficult, or even impossible, to recognise; sometimes, no doubt, because the symptoms themselves are very trivial, frequently because the complications are of so grave a character as to include and mask them. The latter event takes place particularly when the phlebitis occurs as a part of erysipelas, or of diffuse cellular inflammation, or of other kinds of inflammatory affections; it occurs also when pyæm or even sometimes when embolia, supervenes on phlebitis.

But little need be said in reference to the treatment of uncomp cated phlebitis. If the vein be within reach, leeches, or ice (enclos in a bladder or india-rubber bag) may, in the early stage of the affe tion, be applied along its course. At a later stage poultices or war water dressing may be serviceable. Rest should of course be enjoine The constitutional treatment must be made to depend partly on th character and degree of the constitutional symptoms due to the di ease, partly on the patient's general condition of health, partly on th special dangers to be apprehended and guarded against. If the gener symptoms be trivial, little or no medical treatment is called for; bu the more they assume a typhoid character, the more stimulants an nourishment, and medicines tending to the same end as these, ar required. If the patient be suffering from tubercle, cancer, or hear disease; from the syphilitic or alcoholic or other cachexy; or from the effects of privation, the constitutional treatment must have special reference to these conditions, to the patient's general condition of health, and to the special dangers to be apprehended and guarded against.

(2.) *Degeneration.*—Degenerative changes in veins are infinitely more rare than they are in arteries. Indeed, ordinary atheroma is probably never met with here; although it would seem that some degree of fatty degeneration, or at least of deposition of oily molecules, is not unfrequently to be seen microscopically in the walls of varicose veins, and in their valves. Depositions of earthy matter, although very rare, are yet, undoubtedly, of occasional occurrence. And it is worthy of remark that one of their favourite seats is the walls of varicose vessels; those vessels in fact in which fatty matter is now and then discovered. The calcareous deposits form irregular plates (originating apparently in or beneath the lining membrane) almost always considerably thinner than the calcareous plates in arteries, and often presenting a nodulated or "stalactitic" character towards the channel of the vein on which they tend to encroach. These calcareous plates have the same composition, and doubtless the same mode of development, as those in arteries. They consist mainly of carbonate and phosphate of lime, are deposited in globular masses which tend to coalesce, are formed in fact much as bone is formed, and are sometimes converted into unmistakeable bone. They are usually observed in small amount, and often a single calcareous mass alone is discovered.

(3.) *Concretions.*—Phlebolithes are globular or ovoid or irregular concretions, not unfrequently observed in the interior of veins, especially in the interior of veins which are dilated or varicose. Their most common seat probably is in the veins about the neck of the bladder, and other pelvic veins; they are met with, however, occasionally in the varicose veins of the lower extremities, and in the veins of the lungs and of the spleen. They are sometimes attached at one or more points to the lining membrane, are sometimes enclosed in a capsule formed by the obliteration of the vein above, and below the concretion, and sometimes lie loose either in the channel of the vein, or in a pouch connected with it. These bodies vary from the size of a horse-bean downwards, but are sometimes considerably larger. They are of a yellowish-white colour, hard and calcareous, and on section appear to be made up of concentric layers like a urinary calculus. They consist chemically of carbonate and phosphate of lime, with some magnesia, and a variable proportion of organic material. How these bodies are formed has been matter of dispute. By some writers it was imagined they were formed externally to the lining membrane of the vein, into which they subsequently became prominent, then pedunculated, and finally detached. The more common opinion, however, and that which is doubtless the correct one, is that they originate in transformed clots. And, indeed, according to Rokitansky, there is commonly a roundish cavity or irregular fissure within the nucleus, which is itself dry and of a rusty brown or dull yellow colour. A clot of which this nucleus is the remnant is probably first formed; this undergoes degeneration, collapses, and furnishes a nucleus around which fibrinous layers from the blood are successively and slowly formed, and in which calcareous salts become deposited.

(4.) *Adventitious Growths.*—Veins, like arteries, may be involved in carcinomatous and other growths, and like arteries, may become obliterated in consequence. But veins are additionally liable to become the actual seat of such growths. Thus, sometimes when a vein gets surrounded by a malignant tumour, this gradually invades its walls, and ultimately projects into it, and then either forms pedunculated or sessile outgrowths, or fills the channel, renders it impervious, and extends along it. Sometimes, again, secondary cancerous growths originate in the substance of the venous walls, and ultimately form polypi, hanging into the cavity of the vein. We have seen a good example of this condition, in connection with a trunk of one of the pulmonary veins, in a case where the lung was the seat of carcinoma. The same thing may occur also in connection with tumours which are not malignant. Thus in a case of myeloid disease of the humerus, we once found the veins ramifying in the deltoid muscle, and some of the large veins of the upper arm, filled with myeloid growth, identical with that which formed the bulk of the tumour, and continuous with it. Tubercle, so far as we know, is never met with in the veins.

(5.) *Changes of Dimension.*—Veins undergo changes of dimension both from physiological and from pathological causes. To the former class of causes are to be attributed that ordinary enlargement of the veins which attends the growth of the body, that enlargement which takes place in anastomotic branches when a trunk is obstructed, those changes of dimension of the uterine veins which attend the cor sponding change of dimension of the uterus itself, that diminution size which occurs after amputation in the veins of stumps, and so o To the latter class of causes must be assigned the varicose condition veins which is so often met with, and some forms also of contraction veins.

(*a*) *Enlargement.*—Varicose veins are veins which have undergo irregular dilatation. Veins thus affected are elongated and unnatu rally tortuous; their diameter is larger than natural, and often ver considerably larger; and at the same time they present, irregularl distributed over their surface, hemispherical dilatations or aneurism like pouches, and occasionally even flask-like diverticula communicating with them by a comparatively small orifice. In vein furnished with valves the dilatations occur more particularly in connection with the sinuses immediately above them. Coincidently wit this dilatation, the walls become attenuated, and the valves (at firs perhaps simply rendered inefficient by becoming too widely separated from one another) become atrophied and shrivel up. The middle coat of veins is probably that (like the middle coat of arteries) by which dilatation is in the normal condition chiefly opposed; and it is therefore in connection specially with the yielding of this that dilatation occurs. Its fibres become divaricated; and, as already pointed out, they undergo some degenerative process indicated by the deposition in them of fatty particles. Sometimes, on the other hand, the walls of the dilated veins become thickened instead of attenuated, a change which is due to thickening of the outer coat. Dilated veins, like dilated arteries, may, by their pressure on parts external to them, cause the absorption of these parts. In this way the enlarging vessels sometimes approach the surface of the skin, or that of some of the mucous membranes, and even cause the absorption of bone. We have pointed out already that varicose veins are specially liable to become inflamed; they are specially liable also to have coagula deposited within them. These may occur as casts of veins, blocking them up; or may be produced in the dilatations only; sometimes, according to Rokitansky, laminated coagula like those of aneurisms are formed within the pouches. Phlebolithes, as we have already pointed out, are sometimes found in varicose veins, and are probably derived from such clots as have just been described. The varicose condition affects veins very variously, both in extent, in degree, and as regards the order of veins affected. Sometimes the larger veins only are thus dilated, and when such veins are seated in some superficial part, large, tortuous, soft, knotty, bluish cords, projecting above the normal level of the skin, indicate their presence. Sometimes the smaller veins only are varicose, and then, if superficial, they form in different situations pencils as it were of reddish or bluish vessels, larger than natural, thickly clustered and radiating, it may be, from a point or line. Sometimes one or two veins only, or portions of them, present the varicose condition; sometimes nearly all the veins of a limb may be involved in the disease; sometimes there seems a still more general tendency for the veins to become dilated.

The essential cause of the dilatation of veins is the same in principle as that of the dilatation of arteries, namely, inability of their walls, from deficiency in them of resisting power, to withstand the pressure which the blood within them exercises upon them. This inability may depend either on actual loss of power in the walls, or on loss of power due to their over-distension, in consequence of impediment to the onward flow of blood. It may depend therefore on constitutional causes, or on accidental local conditions of disease. But there is an additional circumstance, which has a very important influence in determining the formation of varices, and in increasing their bulk when once they have begun, that is, the pressure to which veins are exposed in relation to the height of the column of blood they have to support. It is, we need scarcely say, a well-known hydrostatical fact, that the pressure exerted by a fluid (whether circulating or still) against any point in the walls of a receptacle containing it, is in exact proportion to the height of the column of fluid above that point; and hence it is clear that those portions of the venous system, which are most dependent or nearest the ground, are exposed to greater pressure from within than those which occupy a higher situation. No doubt this is to a great extent counteracted, in those veins which from their position are most subject to its operation, by the presence in them of valves. Still, it is not wholly counteracted even in healthy veins; and in those which have become sufficiently dilated to render the valves within them useless, it must act to its fullest extent. It is, doubtless, owing in great measure to the operation of this cause that varicose veins are specially frequent, and become specially large, in the lower extremities.

Varicose veins sometimes get well spontaneously, especially after the disappearance of the cause which has induced them. Sometimes a cure is effected by the gradual return of the enlarged veins to their normal size, sometimes by an attack of inflammation in them leading to the deposition of a clot, and to their obstruction and subsequent obliteration. Sometimes they remain more or less stationary. But more commonly, if left alone, they continue to enlarge, and tend ultimately to burst externally and to cause dangerous, even fatal, hæmorrhage. The presence of varicose veins leads also to unhealthy conditions of the parts with which they are connected.

The symptoms due to varicose veins may be gathered from the foregoing account of their morbid anatomy. They consist locally of enlargement of the veins, swelling and œdema of the associated tissues, aching pains, sometimes itching, tendency to inflammation of surface, eczema, excoriation and ulceration. To these may be added special symptoms, due to the impairment of function of any organ or part with which the varicose vessels happen to be connected.

It is not easy to lay down any general rules with regard to the treatment of this affection. If it arise in constitutional condition there is reason of course to suppose that constitutional treatme may be of service. Looking upon the disease then as a diseas indicating debility, it is natural to assume that tonics and other remedies tending to give strength may prove serviceable. And in th present state of our knowledge it is no doubt wisest to act on thi assumption. If, on the other hand, it depend on any local impedimen to the flow of blood, it will be right, if possible, to remove or counterac this local condition. Further, it is generally desirable to support th affected veins, by the application over them of uniform and moderat pressure, in order to obviate the debility of their walls; and, a far as possible, to maintain the part affected in either the horizonta position, or some other position tending to relieve the vessels from undue pressure of their contents.

Before leaving the subject of varicose veins, it is desirable to say a few words in regard to some of the situations in which they chiefly occur, or in which their occurrence is specially interesting. The most common seat of varicose veins is doubtless the lower extremity. Both legs seem to be equally liable to be affected. The disease here presents great varieties; sometimes a small portion of a vein, or a small group of veins only is affected; sometimes nearly all the veins are involved; and all varieties are met with between these extremes. There seems little doubt that the superficial veins are those which, as a rule, are primarily and principally affected; but it has been pointed out by Briquet and by Callender that the points of chief distension are those in which the superficial veins are joined by branches from the muscular and other subjacent tissues; it has also been pointed out that in many cases the deeper-seated veins are equally involved with the superficial veins, and that occasionally the disease is actually limited to those which are deep-seated. Among the conditions tending to produce varicose veins here, and especially, of course, tending to produce them in such persons as are constitutionally predisposed to their occurrence, may be enumerated, occupations requiring long continued maintenance of the erect position, pregnancy, ovarian diseases, and generally the presence of abdominal tumours producing pressure either on the vena cava or on the iliac veins, and probably also cardiac and other diseases in which the free passage of blood from the right to the left side of the heart is impeded. Varicose veins in the leg produce swelling of the leg and sometimes a slight degree of anasarca; they are apt also to lead to inflammatory conditions here, to induration of the cellular tissue, to congestion of the surface, excoriation and eczema, and not unfrequently to ulcers. These ulcers are generally very intractable. It is in the leg, too, more than anywhere else, that rupture of the dilated veins is liable to occur. In the treatment of varicose veins of the leg the constant use of support is very essential. Generally the constant wearing of an evenly applied bandage from the foot to the upper part of the thigh, or of a well-fitting laced stocking, is indicated. Sometimes the obliteration of the

ations as are applicable to superficial inflammation should
ployed. In the treatment of varicose ulcers support and pressure
f the first importance. If a vein burst, the wound should be
d exactly like the wound after the ordinary operation of phle-
ny. The patient, too, should be placed in the horizontal position
e limb elevated.

icose veins not unfrequently arise in the spermatic cord, pro-
g the disease termed varicocele. This occurs almost always upon
eft side, and is supposed to be determined here in part by the
length of the left spermatic vein, and in part by the fact that
vessel opens into the renal vein instead of opening like its fellow
tly into the cava. The veins in this disease become very large
tortuous, and are described as feeling like a bundle of worms;
esticle to which they belong becomes the seat of much aching
, and ultimately its nutrition becomes impaired, it shrinks in size
undergoes atrophy. In this affection the testicle should be sup-
ed; and, as in the former case, it may be necessary to employ
ative measures.

æmorrhoids or piles have generally been regarded as a varicose
lition of the hæmorrhoidal veins; and there is no doubt that they
often produced, and always increased, by constipation and by any
r condition which impedes the passage of blood along these veins,
the veins into which these empty themselves. Hæmorrhoids,
vever, are not so much varicose veins as they are a hypertrophic
dition of the mucous membrane, or of the integuments, in the
ghbourhood of the anus, attended with much congestion of the
llary and other minute vessels, and in some degree also with a
icose condition of the veins.

Varicose veins may occur in other situations besides those which
ve been specified, and lead to grave results. Thus, there is reason
believe that the veins of the stomach and of other portions of the

already specially pointed out some cases of this kind, and we may add to the list, the occurrence of such veins in the abdominal parietes in certain cases of hepatic or of splenic disease, and the dilatation of veins in the neck and upper extremities which follows upon obliteration of the innominate veins, or vena cava descendens, produced by aneurismal or other tumours in the neck. Rokitansky says that the veins of the pia-mater become varicose in drunkards.

(*b*) *Occlusion.*—Occlusion of veins has been already considered incidentally in various parts of the foregoing account of the diseases of veins. It has been shown to occur sometimes as the result of phlebitis attended with the formation of clots; to be produced sometimes by the pressure of a tumour growing external to the vein, sometimes by the growth of carcinomatous or other tumours into the interior of the veins. The results of occlusion and its symptoms have also been considered incidentally. They are principally, dilatation of the veins on the distal side of the seat of obstruction, enlargement of anastomatic veins, œdema of the tissues through which the passage of blood is obstructed, and such further phenomena as attend, on the one hand anasarca, on the other hand varicose veins.

CARDIAC CONCRETIONS.

By John Syer Bristowe, M.D., F.R.C.P.

The condition of the blood in the heart's cavities, at the time of death, as to quantity and quality, and the relation which its varieties of condition bear to the cause of death, are necessarily matters of much pathological interest; they are matters also of some practical interest; and, in both these points of view, have been made of late years the subject of a good deal of careful observation. Yet it is curious that nearly all systematic writers on heart-diseases—and even the more recent of them—have either passed this subject over in almost complete silence, or, if they have been tempted to enlarge upon it, have displayed a lack of knowledge in regard to it which the character of their works in other respects would scarcely have permitted us to suspect.

It is not proposed, in the limited space which has been necessarily allotted to the present article, to treat exhaustively the subject under consideration, still less to criticize at any length published opinions upon it which seem to us erroneous. It is intended rather to give a general brief account of the whole subject, and to enlarge upon those points only which seem to have some special interest and importance.

Morbid Anatomy.—At the time of *post-mortem* examination, the cavities of the heart may be found either contracted and empty, or dilated and containing an amount of blood proportionate to their dilatation. And, in the latter case, the blood may be found either quite fluid, or imperfectly coagulated, or coagulated and moulded to the surfaces with which it is in contact, or in the form of "globular concretions," or in a tough laminated condition, or mixed, it may be, with concretions (emboli) brought hither from remote parts of the vascular system. It may be added, that two or more of the above conditions frequently co-exist in the same case, and such of them as are not incompatible with one another, even in the same cavity. It is desirable, however, to discuss them separately.

Emptiness of Cavities.—The cavities which are most frequen found empty of blood are the ventricles. This emptiness is mu more common in the left ventricle than in the right; but not frequently both cavities are in the same condition. The auricles generally full, if not distended.

Fluid and semi-fluid Blood.—The blood contained in the hear cavities may be fluid or semi-fluid. That is to say, it may be nea as fluid as when it freshly escapes from a vein, it may be more less treacly, or it may contain floating in it soft loose masses of dar coloured imperfectly-formed clot. It is in these cases usually th the lining membrane of the heart and large vessels becomes staine with the colouring matter of the blood.

Moulded Clots.—The blood may have undergone more or less com plete coagulation. Its condition, however, varies very considerably i different cases. Sometimes the coagula are small, and the cardia parietes are contracted, or collapsed upon them; sometimes they ar large and distend the cavities to the full; sometimes they exis therein alone; sometimes they are surrounded by a greater or les quantity of serum or of uncoagulated blood; sometimes they are o a uniform red-black hue; sometimes they are partly decolourized; sometimes they are wholly fibrinous.

These coagula, whatever their colour or consistence, are always accurate, or nearly accurate, casts of the cavities which contain them, and are generally attached to the surface, not by any organic connection, but by being dove-tailed, as it were, with its inequalities. Those of the corresponding auricle and ventricle are continuous through the auriculo-ventricular opening; and are, moreover, prolonged to a greater or less extent into the venous and arterial trunks. In the aorta they sometimes extend throughout nearly its whole length, in the pulmonary artery to its smallest subdivisions. The prolongations into these tubes are cylindrical, but generally a good deal smaller in diameter than the tubes themselves; and those portions of them which correspond to the arterial valves, have always the form of the valves distinctly impressed upon them.

Moulded coagula are sometimes, as has been just pointed out, of a uniform red-black hue. They have then much the appearance and consistence of black-currant jelly, are soft and tremulous, and consist of a uniform mixture of chiefly the fibrine and the red corpuscles of the blood. Sometimes they are partly decolourized, or, in other words, a partial separation of their component elements has taken place. The fibrine may then have separated, much as it does in the formation of the buffy-coat after bleeding, producing a thin almost colourless layer on that portion of the clot which, during its formation, has lain uppermost. Or it may happen that the whole surface of the clot is fibrinous, while the interior remains coloured. Sometimes again, and this is the most remarkable case, the whole, or nearly the whole, of the clot is fibrinous. Such clots are sometimes loose in texture and watery, and retain more or less of the colouring matter of the blood;

sometimes straw-coloured, jelly-like, and elastic; sometimes close-grained, buff-coloured, opaque, and tough.

Moulded clots may be found in all the cavities of the heart: but those which are fibrinous are chiefly found in the ventricles, and more frequently in the right ventricle than in the left; they may, however, occur in the left ventricle even when they are absent from the right. The clots which extend into the larger vessels are generally in the greater part of their extent identical in character with the cardiac clots, with which they are continuous. But even when almost purely fibrinous, they mostly pass off at their extremities into coloured clots. This is especially the case with those occurring in the veins.

Softening Clots.—Softening clots, globular concretions, purulent cysts (for all these names, and many others, have been applied to the bodies now about to be described) are coagula, which have undergone changes, by which they have become converted into roundish masses, softened for the most part internally into a puriform fluid, attached firmly to the parietes, and occupying, with scarcely an exception, those portions of the cavities which lie out of the direct current of the blood.

These bodies may occur singly in a cardiac cavity, or in considerable numbers, and may vary from the size of a pin's head up to that of a pigeon's egg. They are almost always attached to the surface, and generally spring distinctly from the interspaces between the carneæ columnæ, or the musculi pectenati. Their attachment to the surface, though sometimes in part due to slight adhesions, is mainly effected by this entanglement with the fleshy columns; and, indeed, where several of these bodies are present in a cavity, they are probably always continuous with one another by means of processes extending beneath those muscular bands which are attached to the cardiac walls by their extremities only.

Their free surfaces are sometimes smooth, sometimes more or less ribbed; generally they have an opaque buff-colour, but they may present more or less of a brick-red tint, or may be variegated with irregular streaks of red and white. On section, they present considerable variety of appearance. Sometimes they are solid throughout, and repeat on their sectional surface, the characters already displayed by their external aspect; more commonly, however, they are more or less softened, at one time converted into a thin-walled cyst, at another time broken up irregularly into a series of small intercommunicating cavities. The walls of the cysts are identical in character with the substance of the unsoftened concretions, but their inner surface is soft and flocculent. The contained fluid is thick and puriform, and varies in colour from a pale buff to a brick-red, or even chocolate, hue.

Under the microscope the solid portions of these concretions are found to consist of a fibroid network similar to that of ordinary coagulated fibrine. "This, however, is intermixed with a large quantity of granular matter, which renders the fibroid structure more or less indistinct. They contain also oil, compound granular cells, and a few imperfect cells which appear to be the remains of whit corpuscles. In some cases there are many altered blood-corpuscle and now and then solitary and clustered needle-like crystals." "Th puriform contents of the cysts present considerable variety as to thei microscopic elements. When white or buff-coloured, they consis almost solely, if not solely, of molecular matter, oil, and broken down corpuscles, with which are frequently mixed compound granula cells, and colourless acicular crystals. When presenting a brick-red o chocolate hue, they exhibit, in addition to the elements just mentioned, numerous blood-corpuscles, more or less altered, and consequently more or less indistinct, and occasionally also ruby-coloured rhomboidal, hæmatoid crystals." In one instance which we hav met with, the fluid contents consisted almost entirely of well-marke pus-like corpuscles.

It is asserted by Rokitansky,[1] that these concretions are almos always limited to the left ventricle. This, however, is an error. They do, it is true, occur here more frequently than in any other one of the cavities of the heart; but they occur much more frequently in all the other cavities collectively, than they do in the left ventricle. They are not unfrequently found in two or three cavities, and occasionally in all of them at the same time. In order of frequency they affect, we believe, first, the left ventricle; second, the right ventricle; third, the right auricle; and last, the left auricle. With regard to their position in the cavities of the heart, there is no doubt that, with scarcely an exception, they occupy those situations which are most favourable to the stagnation of blood. "In the auricles they chiefly affect the auricular appendages, and in the ventricles they almost always occupy the spaces and interstices between the carneæ columnæ."[2] Occasionally, they are developed around some of the chordæ tendineæ; and one or two cases are recorded, in which they have been found detached.

Laminated Clots.—Laminated coagula, such as are found in aneurisms, are of very unfrequent occurrence in the heart, and of recorded cases, the most common are certainly those in which the coagula have formed in the interior of aneurismal dilatations, or of actual aneurisms developed in connection with the ventricles. Still a small number of cases have been met with, in which cavities otherwise healthy have become almost obliterated with coagula of this kind. We are acquainted with this condition only as affecting the left auricle, secondarily, to extreme contraction of the mitral orifice—under circumstances, therefore, not dissimilar from those which lead to the formation of such coagula in the interior of actual aneurisms. In one such case,[3] where the mitral orifice was so contracted as scarcely to admit the tip of the little finger, the left auricle was greatly dilated, and full of firm laminated coagulum, which formed two perfectly distinct masses—one extending from the auricular appendage backwards, the other forwards from the posterior and inner part of the cavity. They were slightly adherent to the parietes, and were in contact with one another by their free surfaces, which were consequently flattened. The cavity of the auricle was thus obliterated, or, at least, reduced to the imperfect and irregular channel left between these mutually compressed masses.

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[1] Path. Anatomy (Sydenham Society's Translation), vol. iv. p. 217.

[2] The passages included within inverted commas are quoted from the author's papers, "On Softening Clots in the Heart," contained in the 7th and 14th volumes of the Pathological Society's Transactions.

[3] See Path. Trans. vol. xi. p. 65.

Embolic Concretions.—Sometimes, though very rarely, concretions which have been moulded in remote parts of the vascular system are found entangled amongst ordinary cardiac clots. The only instance in which we have certainly met with this condition was a case of scarlet fever with sloughing of the tonsils. In this case small opaque shreds, and portions of cylinders, consisting entirely of corpuscles resembling those of pus, were found in the right ventricle, embedded in ordinary *post-mortem* clot. We have never met with tubercle or carcinoma in the heart thus conveyed.

ÆTIOLOGY.—It is obvious that at the moment of death the heart's ventricles are either contracted, or in various degrees dilated; and that their emptiness or fulness of blood at the time of *post-mortem* examination must be in great measure determined by these conditions. It is obvious, too, that in those cases in which the cavities are found full of blood from the stagnation in them simply of the blood arrested in its course at the moment of death, the state of this blood as to fluidity or coagulation must depend in great measure upon the conditions of sickness under which death has occurred. All these are matters of interest, and worthy of investigation; but they are not included within the scope of our present article, and we are compelled therefore to dismiss them.

But of the *clots* found in the heart after death, some have evidently been formed in it during life, and may possibly have had some influence in destroying life. Amongst these must be included such as are wholly or for the most part fibrinous, globular concretions, and laminated coagula. How and by what means these are produced we have now to consider.

Clots moulded to the cavities of the heart, if they be of uniform consistence and of a uniform reddish-black colour, have doubtless in all cases been formed *post mortem*, from fluid blood contained in the cavities at the time of death; and the same explanation doubtless holds good of those cases also in which such clots present a layer of fibrine (a buffy coat in fact) on that part of their surface which has lain uppermost. In all cases, however, where the clots are purely fibrinous, or where the fibrinous element is in excess, or where the fibrine which has separated occupies any other position than the upper surface, the separation of the fibrine and therefore the coagulation of the blood, must have taken place during life, while the blood was still in process of circulation. That this must be so is eviden from the consideration that there is no means by which stagnant flui blood can, in coagulating, manifest separation of fibrine except upon its upper surface, still less achieve the perfect separation of its fibrin from all its other constitutents. It is further proved by Dr. Richardson's examination[1] of these fibrinous clots, which shows that th amount of fibrine contained in them is several times greater than can be accounted for by the quantity of blood which the heart's cavitie are capable of containing. It must not be forgotten, however, that al the fibrine met with in such cases in the cavities of the heart rarely if ever, exceeds the amount of fibrine contained in the blood which passes through the heart in the course of half a dozen beats; and that therefore, the whole of a large fibrinous clot may have been whipped out of the blood in the course of the minute or two of circulation which precedes death. It is certain, then, that such clots are formed during life, but by no means clear how long their formation actually takes. Some of them are doubtless, as has been just suggested, formed in the course of the few moments immediately preceding death; but it is exceedingly probable that others have taken some considerable time in their formation.

What it is that determines this coagulation of the blood during life is by no means easy to determine. Dr. Richardson, in the paper before referred to, enumerates several classes of cases in which fibrine is peculiarly apt to be deposited during life in the heart's cavities, the most important of his classes being that of acute inflammatory affections, including pneumonia. We have no doubt that, in all the cases which he enumerates, fibrinous clots are not unfrequently observed; but, indeed, they are constantly met with in the *post-mortem* room, not only in them, but in almost every form of disease. They are by no means constant, even in cases of pneumonia. Mr. Henry Lee believes them to be characteristic of purulent infection of the blood This, however, is obviously an error; for while it is common to meet with them in cases where no such infection can be suspected, in cases of pyæmia they are altogether exceptional. We shall not pretend to offer any satisfactory explanation of the causes of the formation of these *ante-mortem* clots in some cases, and their non-formation in others. But we may admit generally with Dr. Richardson, that there are diseases in which, from some cause or other, there exists a tendency to the separation of fibrine; and further we may suggest that slowness in dying may in such cases to some extent determine this separation..

With regard to the formation of the rounded concretions, which are generally softened in their interior into a puriform fluid, many fanciful theories have prevailed. Thus it has been supposed that they are softened tubercle, or pus, conveyed to the heart from a distance and there encysted. Their contents, however, are never tubercular, and rarely if ever purulent; and although they may be occasionally met with both in phthisis and in pyæmia, their occurrence in these diseases, especially in the latter of them, is exceptional. Again, it has been supposed that their formation is due to local endocarditis. But in reply to this supposition, it may be pointed out that they rarely, if ever, accompany undoubted cases of endocarditis; and, moreover, that they are almost without exception found in just those parts of the heart's cavities in which true endocarditic deposits probably never take place. That they are merely altered clots is evident, both from their microscopical constitution and from their identity, in the changes which they undergo, with clots formed in other parts of the body, whether in the vessels or by extravasation. It is evident, too, that the condition in which they are found after death is the result of processes which must have required days, or even weeks, and possibly a still longer time for their completion. It is evident, further, from the mode in which they are attached to the cardiac walls, that they must have been formed in the position in which they are discovered *post mortem*. The cases in which they are most commonly observed are cases of heart disease, of renal disease with dropsy, of chronic bronchitis, and of chronic phthisis, cases in which death is often slow, or in which struggles, as it were, between life and death are apt to occur from time to time for some while before death actually supervenes. It seems probable that the foundation of these concretions is laid at one or other of these moments of seemingly impending death, by the coagulation at that time of blood in the cavities of the heart; that the patient rallies from his apparently moribund condition, and that the clots, at once the evidence and the result of that condition, remain; that the clots then during the remainder of the patient's life gradually undergo those changes, which clots in the brain and elsewhere are liable to undergo; that they become torn into smaller masses, probably in consequence of the constant movements of the cardiac walls; that these masses become rounded partly in consequence of the contractile force inherent in the fibrine of which they chiefly consist, partly by the attrition to which they are exposed by the constant movement of the fluid blood over their surface, and that after a while their interior undergoes softening and disintegration.

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[1] See Dr. Richardson's Lectures in the British Medical Journal for 1860.

The laminated clots, of which I have quoted an example from the left auricle, are evidently of slow growth, and originate long anterior to death. Indeed, they are obviously formed on the same principle as that which determines their formation in aneurisms, and are the result, as in aneurisms, of a slow process of deposition from the blood.

SYMPTOMS AND EFFECTS.—We now have to consider the important question, whether the clots which are formed in the heart prior to death have any effect in producing death, and if so, whether their presence during life can be recognised by any characteristic symptoms. All who have enjoyed much clinical, combined with *post mortem*, experience of disease, will admit, as regards the vast majority of cases in which *moulded fibrinous clots* are discovered in the heart, that their formation has taken place during the process of dissoluti and as a part of that process, that their formation has been unattend with symptoms referable to themselves, and that if they have exert any influence adverse to life, it can only have been in the sen of preventing any tendency to rally, in other words, of confirmin the act of dying. It does not however necessarily follow th cases do not occasionally happen, in which in the course of certai forms of disease, or even apparently in health, such clots form, an by the impediment which they oppose to the circulation of the bloo through the heart, cause death. To the consideration of this poin we will shortly recur. Meanwhile we will discuss the effects of thos forms of clot—*globular and laminated concretions*—which beyond al dispute must have been in existence a considerable time anterior to death. In regard to the laminated concretions, it may be stated, we think with some degree of certainty, that their presence is attended with no special symptoms. No doubt they add to the embarrassment of the heart, but they add only to the embarrassment of an already embarrassed organ; they merely increase the severity of symptoms which are already severe, and therefore, if combined with mortal disease, merely cause the disease to anticipate its final series of events. It is worthy of remark, however, that life is maintained in these cases even when the auricular cavity is so encroached on as to be no more than a mere channel between the veins and the auriculo-ventricular opening. Globular concretions equally as a rule produce no special symptoms. Certainly they are constantly met with *post mortem* in cases which have been under continued observation, and have presented no special symptoms indicative either of their formation or of their presence. No doubt their presence in the cavities of the heart has a tendency on the whole to impede the action of the heart and to affect the circulation injuriously, especially if they be large, or if they occupy certain situations. But impediment, real or virtual, to the circulation, probably always exists prior to the formation of these bodies, so that their addition tends to aggravate symptoms already established rather than to develop new ones. It seems not improbable that they may now and then interfere with the normal function of some of the valves, and so lead to the production of endocardial murmurs, but with this result we have no practical acquaintance. Again, two or three cases are recorded in which they have been found detached in an auricle, and lodged in, and thus obstructing, a contracted auriculo-ventricular orifice. And it has been surmised that they may occasionally become ruptured, and, by the escape of their contents into the circulating blood, produce symptoms of pyæmia. It may be considered, therefore, that, excluding a small number of quite exceptional cases, the presence of these clots in the heart cannot be recognised by peculiar symptoms, but may be surmised, and often correctly, in cases where the struggle between life and death has been greatly protracted, especially if the patients be suffering from any of the diseases in which morbid anatomy shows that these clots are chiefly produced. It is important, however, to bear in mind that although these concretions doubtless originate in ordinary fibrinous coagula, there are few if any cases in which the moment in which they were first formed can be even approximately determined.

Let us now return to the question as to the influence of moulded clots in producing death. It seems to us that with the facts before us—first, that coagula of this kind are constantly observed in the *post-mortem* room as the mere accompaniment and result of the dying process; second, that (as has been shown) whole cavities may become obliterated by coagula without directly causing death; third, that (as has also been shown) in the majority of cases in which it can be clearly demonstrated that concretions have been formed some considerable time before death, their formation has not produced marked symptoms, we ought to require very strong testimony indeed to convince us in any case that concretions found in the heart at the time of death, have caused death, still more to convince us that those clots which resemble in every point the clots which are the mere result of dying, have had this effect. It is no doubt convenient and seductive, when we meet with a case of fatal illness, to be able to point to some obvious pathological phenomenon attending it, and to believe that in that we recognise the cause of death. Not long ago fatty heart furnished the ready explanation of most sudden deaths, now fibrinous concretions in that organ begin to rival fatty heart in popularity. We have no hesitation in stating our conviction that in the great majority of cases which have been recorded of death from the formation of fibrinous concretions in the heart, these concretions have been developed in the ordinary way, and have had no more to do with the death of the patient than the rigor mortis has. We are not prepared to deny that death is sometimes actually caused by such a deposition of fibrine, but we can state positively that no such case has come under our observation, and we believe that the great majority of recorded cases are cases in which the sequence of events—cause and effect—have been misunderstood and transposed. In the remarks which have just been made, we wish it to be distinctly understood that we refer exclusively to cardiac concretions, and not to concretions blocking up the pulmonary artery and limited to that artery. This latter subject will be discussed under the head of "Thrombosis and Embolia." We may add, for the convenience of those readers who are interested in the subject, that they will find an ingenious account of the symptoms which are supposed to attend the formation of moulded cardiac concretions, in Dr. Richardson's lectures, already more than once referred to in the course of this article.

THROMBOSIS AND EMBOLIA.

BY JOHN SYER BRISTOWE, M.D., F.R.C.P.

THE terms "Thrombosis" and "Embolia" (or Embolism) have been introduced by Virchow: the former, to signify the coagulation of blood in arteries or veins during life: the latter, to signify the transference either of clots, or of other solid matters appearing within the vascular system, from one part of that system to another part, in the direction of the circulating current, and by means of it. These subjects have already been partly considered under the heads of "Pyæmia," "Cardiac Concretions," "Diseases of Arteries," and 'Diseases of Veins;" we propose however here to treat them as a whole, and, as they are intimately related to one another, to combine their description in a single article.

The local phenomena, which attend the coagulation of blood in the vascular system during life, are essentially the same, in whatever part of that system coagulation takes place; and the changes which clots undergo are also essentially the same, whether the clots occur in the arteries, in the veins, or even in the heart. These have been already in great part described. A clot, consisting either of nearly pure fibrine or of all the solid elements of the blood combined, forms, and is moulded as it forms, to the surface against which it lies; to which also it is from the beginning, or becomes ere long, adherent. The changes which such a clot undergoes in the course of time vary. They consist, sometimes in its gradual contraction and organization; the fluid matters become absorbed, the cellular elements disintegrate and disappear, the fibrinous portion undergoes condensation, and ultimately the clot becomes converted into, or replaced by, ordinary connective tissue. They consist sometimes in the softening and breaking down of the clot internally; the central parts become converted into a thick puriform fluid, sometimes red, sometimes nearly white, consisting chiefly of disintegrated cell elements merely—such as granular matter, oil, cholesterine, debris of corpuscles, and perhaps hæmatoid crystals; the clot may thus come to form, either wholly or in part, a mere fluid-holding bag, in which condition it may remain for a considerable time; but gradually, here as in the former case, the fluid portion undergoes absorption, the contents dry up and the cyst-walls collapse upon them. Sometimes, further, clots become the seat of calcareous transformation; and this may occur both in those which have softened internally and in those which have maintained the solid form; particles of earthy matter are deposited, which gradually increase in number, and ultimately by their aggregation transform them into calcareous masses. There is reason, as has been already shown, to believe that phlebolithes are formed in this way; and phlebolithes are occasionally the seat of true ossification.

A full account of these clots, as they are met with in the heart's cavities, has already been given.

In the aorta and pulmonary trunk they are unfrequent, except where they are met with as fibrinous or more or less coloured cylinders prolonged from the interior of the respective ventricles or from the neighbourhood of the semilunar valves. Such clots, like the corresponding cardiac clots, are manifestly formed during life, though often during the last moments only of life, they always present the impress of the arterial valves, and, though generally much smaller than the channel in which they lie, sometimes almost fill it. Older clots are sometimes observed in the aorta. These are isolated roundish concretions, adherent to the surface (mostly if not always in connection with points of disease), projecting into the canal, but not materially obstructing it. In other arteries, however, and especially in the smaller arteries, these clots generally form solid cylinders, equal in diameter to the vessel in which they lie, adherent more or less to its surface, and more or less completely obstructing its channel. The obstruction, however, is generally at first incomplete, and the constant impulse of blood against the proximal extremity of the clot tends gradually to force a certain proportion of blood between it and the arterial walls. In this way, blood in small quantities flows for a time through irregular channels over the surface of the clot; soon, however, it coagulates there, and thus the original clot becomes incrusted with an irregular layer of more recent coagulum, the vessel becomes distended and the occlusion becomes complete. Further, additional coagulum tends to be deposited in connection with the extremities of the primary clot; this deposition ceasing generally, on the proximal side, at the point of anastomosis nearest the seat of obstruction.

The brief account which has just been given, applies with almost equal exactness to the clots which form in veins. A very important additional fact, however, in regard to these, has been demonstrated by Virchow; the fact, namely, that they tend to increase by the deposition of fresh clot at their proximal end, until the vein becomes filled up as far as its mouth, and that, in continuation of this process (from the blood which passes along the trunk vein with which the obstructed vein communicates), additional coagulum is gradually added to that which has been already deposited, until from the plugged orifice there projects into the interior of the trunk a rounded mass of laminated coagulum, which may attain a very considerable size.

The causes of Thrombosis have already been to some extent considered. Sometimes the coagulation seems to be consecutive to mere stagnation of blood, or sluggishness of circulation, occurring in certain conditions of disease. Such probably is the case in regard to softening clots in the heart; such probably, also, is the case in regard to the clots which plug certain of the veins in phthisis and some other affections; and such, also, doubtless, is the case when arteries, leading to districts of disease in which the capillaries are obstructed, become themselves filled with clot. Sometimes the coagulation is determined by mere roughness of the surface over which the blood passes. This is observed when isolated clots become adherent to atheromatous patches, and when extensively atheromatous or ossified arteries become obstructed with clots. Sometimes the thrombus is the result of inflammation of the walls of the artery or vein in which it is found, the coagulation of the blood being consequent on some altered relation between the walls of the vessel and the blood within them. Indeed, phlebitis and arteritis are probably the most frequent causes of thrombosis. Further, the formation of clots in arteries and veins takes place occasionally in the course of some cachectic conditions of system, such as those connected with syphilis or anæmia. It may, of course, be a question, whether or not the coagulation in these cases even may not be the result of inflammation.

The embolus, or obstructing mass, which, conveyed from a distance, becomes lodged in some vessel, and, for the most part, occludes it, may consist of any solid material derived either directly from the blood, or from the walls of certain parts of the vascular system. But in order for Embolia to take place, it is obvious that the solid matter must be formed in such a situation as shall admit, first, of its detachment, second, of its conveyance by means of the circulating fluid, third, of its impaction in some vessel too minute to admit of its further progress onwards. Hence it follows that an embolus must always be looked for in some part of the pulmonary or systemic arterial system, or in the portal system, and that its source must be sought for, as a rule, either in the veins or in the heart; occasionally in the large arterial trunks.

A very frequent source of Embolia is the formation of clots, from whatever cause, in the systemic veins. Thus, sometimes phlebitic or other clots become dislodged, then swept away in mass by the blood, and ultimately fixed in some part of the pulmonary arterial system. More commonly, however, as Virchow has shown, thrombi, which have become friable in texture, undergo disintegration, so that fragments only of them become detached and carried onwards; and he has shown that this process chiefly occurs in connection with those bulbous extremities of thrombi which project from the occluded veins into the trunks with which these veins are connected. In this case the emboli are likely to be numerous and small; and it is likely that many of the smaller twigs of the pulmonary artery will be occluded rather than one or two of the larger branches only. The same processes may take place in connection with the pulmonary veins and systemic arterial tree; but Thrombosis in these veins, and Embolia from this source in the systemic arteries, are certainly not common.

Another frequent source of emboli is furnished by the interior of the heart. Sometimes, there is reason to believe, the softening clots, which have been already described, and which it has been shown may be detached, may be conveyed onwards, and produce arterial obstruction. The most frequent source by far, however, in connection with the heart, is the vegetations which form on the valves in the course of rheumatic and other inflammatory conditions. Sometimes the soft granulations of recent inflammation, which are often clustered, and often loosely attached, become separated and washed away with the current of blood; sometimes, on the other hand, fragments of older concretions break off—concretions which have become condensed and friable, or tough. In either of these cases, it may happen that the detachment of one or two large masses may lead to the blocking up of some arterial trunk, or (and this is more commonly the case) that the separation of a number of small fragments may cause the occlusion of one or many small vessels. Since cardiac vegetations, as the result of inflammation, are much more common on the left side of the heart than on the right side, it necessarily follows that Embolia originating from the heart is much more commonly met with in the systemic arteries than in the arteries of the lungs.

The last source of emboli is the crumbling away or disintegration of atheromatous or cretaceous deposits, such as one meets with in erosion or ulceration of the lining membrane of the heart or arteries. The minute particles, or, at least, some of them, not unfrequently become arrested, like other emboli, in the arterial twigs, and lead to their obstruction. As in the last case, Embolia from this cause is much more frequently observed in the systemic arteries than elsewhere.

An embolus, of whatever kind it may be, and whatever may be its source, becomes swept along with the blood, from one vessel to another vessel, until it reaches one which from its size opposes a bar to its further progress; in this it becomes wedged, and obstructs it wholly, or almost wholly. Very often it becomes fixed on the spur formed by the bifurcation of an artery. Soon after its arrest the embolus becomes invested in clot; blood gradually coagulates on its proximal side as far back as the next anastomosis of the obstructed artery; it coagulates, also, generally on the distal side as well, sometimes only as far as the next branch, sometimes throughout the whole series of vessels which the primarily obstructed artery supplies. In the case of obstruction of vessels from emboli, equally as in that from thrombi, blood will, in a greater or less degree, insinuate itself for a time between the embolus and the vascular wall; this blood, however, generally soon coagulates, and becomes continuous with that at either extremity of the embolus. The clot in which the embolus thus becomes imbedded, and which completes the obstruction which the embolus had begun, may vary in character from an ordinary coloured clot to a purely fibrinous one. At first it is easily distinguishable from the embolus within it but, like other deposited clots, it soon undergoes degenerative change and gradually approximates, more or less, in character to the embolu itself, which may thus be rendered quite incapable of separate recognition.

Generally, at all events in the more obvious cases, the embolus is definite mass, which becomes impacted in the form in which it had separated. Not unfrequently, however, especially when the more minute arterial twigs become obstructed, these twigs are found distended with an aggregation of small angular masses, which would seem to be either the débris of a larger embolus, or the minute particles due to the erosion or crumbling occurring at the seat of the primary affection. It seems not improbable that both of these latter explanations may hold good of certain cases; that sometimes, as Virchow suggests, an embolus which has become impacted breaks up into fragments, under the constant pressure from behind to which it is exposed, and that these fragments become then driven onwards into the minuter vessels beyond; that occasionally also, perhaps the débris, separated from an eroded surface, become in the process of separation loosely cemented together by coagulum, and the soft mass thus formed becomes driven into the minuter arteries, and moulded to them.

The local indications of the presence of a thrombus and of an embolus are as nearly as possible identical. In both cases the vessel becomes obstructed and distended; in both, inflammation of the walls (even if it did not previously exist) becomes excited, and they undergo thickening; and in both, if the vessel affected be superficial, it may be felt to be enlarged or hardened, and will probably be found to be painful and tender. The most important results, however, of these affections are those which depend on the obstruction of vessels—results which manifest themselves in connection with the parts which lie on the distal side of obstruction, and especially in those regions, the vessels of which are tributaries or effluents of those which are obstructed. It has already been pointed out generally what these results are. When a vein is obstructed, the return of blood is prevented in a greater or less degree, the vessels behind become distended with blood, dilated—it may be varicose—and the tissues behind become the seat of congestion and of dropsical effusion. When an artery is the seat of obstruction, the direct passage of blood to and through the parts to which the artery leads becomes arrested. In some cases, of course, this disturbance of the circulation is temporary only; anastomozing branches enlarge, and, by transmitting an increased quantity of blood, make up between them for the loss to the circulation of the obstructed vessel. But in all cases disturbance takes place to some extent; the nutrition of the districts to which the artery leads becomes impaired, the blood stagnates in its vessels, these become distended with blood, owing to the reflux into them from neighbouring vessels, and not unfrequently ruptured so that extravasation takes place; sometimes inflammatory processes, with exudation of lymph, or suppuration, supervene; and very often molecular death and gangrene ensue.

The gravity of the consequences of obstruction of vessels, whether arising from Thrombosis or Embolia, depends partly on the size of the vessel obstructed, partly on the importance to life of the organ or part to which the blocked-up vessel belongs, partly on the suddenness with which occlusion takes place. Obstruction from one or other of these causes may affect any vessel. As regards obstruction of veins, all that might otherwise have needed to be said here has already been said under the head of Phlebitis, from which venous Thrombosis can scarcely be separated. But some of the more important cases of obstruction of arteries we propose now briefly to consider seriatim.

Obstruction of Arteries of Heart, Liver, Spleen and Kidneys.—In each of these cases, what Virchow terms capillary emboli, derived from cardiac granulations, are far from uncommon. Occasionally large masses become impacted in certain of their vessels; and occasionally (in the kidneys especially) without any cardiac disease whatever, the larger number of the principal arteries of the organ become obstructed by the formation of firm fibrinous adherent clots within them. The consequences of obstruction of the arteries of the organs just enumerated are not very dissimilar from pyæmic affections of the same organs; they may consist in all (but more especially in the heart and kidneys) of minute abscesses, about as large perhaps as pins' heads, or of minute abscess-like points, in which the puriform matter is composed of mere disintegrated material; they may consist also in all (but more particularly in the spleen, kidneys, and liver), of so-called "fibrinous blocks." These vary in size, but are often very large—a cubic inch or so in bulk. They present, for the most part, well-defined limits, vary between a pale buff colour and a deep brick-red hue, and appear to consist essentially of the normal tissues infiltrated with some of the elements of blood. The pathological phenomena here described are very frequently observed, but the symptoms to which they give rise are not very obvious.

Obstruction of Arteries of Brain.—Far more important than the obstructions which have just been considered are the obstructions which, as Dr. Kirkes originally showed, take place occasionally in the arteries of the brain. The obstructions here are, without doubt, frequently embolic, and take place distinctly in the course both of chronic diseases of the aortic or mitral valves, and of acute inflammatory attacks of these parts. When such is the case the embolic fragment is almost invariably discovered in one or other of the middle cerebral arteries, or their branches—according to Dr. Kirkes, most commonly in the artery of the right side. But the obstruction also frequently takes place wholly independently of heart disease, and without any possible source of Embolia, and is clearly then due to Thrombosis of the affected vessel. Such Thrombosis may occur in any of the arteries at the base of the brain, and we have seen a case in which the cerebral portions of both internal carotids and the basilar artery became thu successively obstructed. The effects of obstruction of arteries on th portions of brain-substance to which the obstructed arteries lea consist, in the first place, of patchy congestion, and, in the secon place, of marked softening, attended with yellowish, or slightl greenish discoloration, and the appearance of numerous compound granular cells. The affected portions of brain are usually small and circumscribed, but are sometimes extensive and diffused. They are most frequently observed (in connection with obstruction of the middle cerebral artery), in the corpus striatum.

The symptoms of obstruction of the cerebral arteries always appear suddenly. The patient is seized with a kind of "fit," sometimes apparently epileptic, sometimes syncopic, but sometimes unattended with either convulsions or loss of consciousness; and on emergence from this sudden attack he is found to be hemiplegic. The symptoms which succeed are little, if at all, different from those which attend on apoplectic attacks; they vary in different cases, as the latter vary, and need not be detailed in this place.

Obstruction of Arteries of Extremities.—The arteries of the extremities become occasionally obstructed by clot. Sometimes no doubt these obstructions are embolic; but far more commonly we believe they depend on arteritis. In the lower extremity, where this condition is most frequently observed, the seat of obstruction is usually, we believe, either the femoral artery in the neighbourhood of the origin of the profunda, or the popliteal artery. Occasionally obstruction takes place simultaneously in the corresponding arteries of opposite limbs. The formation of a plug is generally, perhaps always, ushered in by acute pain at the spot which the plug occupies. This is followed by impaired circulation in the limb beyond, loss of pulsation in the distal portion of artery, pallor, coldness, numbness, and ultimately, it may be, gangrene. In certain cases, however, the patient recovers from the effect of the occlusion, as patients recover from that produced by the ligature of an artery.

Obstruction of Pulmonary Arteries.—The branches of the pulmonary artery are the recipients of all the emboli derived from the systemic venous system. Embolia, therefore, is in them of common occurrence. Thrombosis also not unfrequently takes place in them. The blocking-up of minute arterial twigs in pyæmia leads, as has been already shown, to the morbid changes in the lungs indicative of that malady. The formation of clots in some of the arteries, in the course of mitral and other forms of obstructive cardiac diseases, are constant accompaniments of pulmonary apoplexy and probably in most cases precede and cause it. Occasionally the impaction of a large embolus in one of the larger branches of the pulmonary artery, or the development therein of a thrombus, leads to inflammatory, and other, mischief of a comparatively large portion of the lung, or even of an entire lobe. We have seen such a case, in which a large portion of lung, the main artery of which was obstructed by an embolus, had become pneumonic, its terminal bronchial tubes had become destroyed by suppuration, and thus converted into irregular cavities, and the investing pleura had become inflamed. The most serious cases, however, are those in which, either from Thrombosis or Embolia, the whole, or nearly the whole, pulmonary circulation becomes suddenly arrested, and rapid or sudden death ensues. A good many cases of this kind are recorded, and they seem to be comparatively frequent among puerperal women, though they are by no means confined to them. Occasionally no doubt the transference of a clot which has formed in one of the systemic veins is the cause of this sudden obstruction, either by the clot itself blocking up the pulmonary trunk, or by serving as a nucleus, around which, after its impaction, further coagulation takes place. Much more frequently, however, we believe, the formation of the obstructing clots commences, and becomes completed, in the arteries in which they are discovered after death. And we ground this belief on the fact that in many cases, where death from this kind of obstruction is unquestionable, the pulmonary clots form an almost continuous system, accurately, or nearly accurately, moulded to the channels in which they are found, and in a greater or less degree adherent to them. It is a very remarkable fact that, in some at least of these cases, the deposition of clots in the pulmonary arteries must have taken place, and been completed to the verge of almost total obstruction during a period of nearly perfect health, and that the patient's sudden death has been due either to an accidental shifting of position of the clot, or to the coagulation of the streamlets of blood, by the persistence of the flow of which between the older clots and the arterial walls life had hitherto been maintained. The clots here referred to commence sometimes immediately above the pulmonic valves, sometimes at the bifurcation of the pulmonary artery, sometimes separately in each branch of the artery, and are continued more or less uniformly, and for a greater or less distance, along their ramifications. They possess all the characters of clots formed some while before death, and adhere here and there to the arterial walls.

The symptoms which indicate serious obstruction of the pulmonary artery are sudden embarrassment of respiration, great dyspnœa, with coldness, and pallor, and clamminess of skin; pallor, not lividity, of face; feebleness, rapidity and irregularity of pulse, followed by death, sometimes after an interval of several days, sometimes quite suddenly. As an example of the formation of clots in the pulmonary arteries, or rather of their presence in these arteries, during apparent health, and of sudden death resulting from their presence, we may quote the following case:—

A female servant, thirty years of age, was admitted into St. Thomas's Hospital under Dr. Bristowe's care, on the 18th June, 1860, having suffered from slight symptoms of pleuritis on the right side for about ten days. On admission there was distinct evidence of dry pleurisy on the affected side, but the symptoms soon passed away;

the patient got apparently well, and was about to leave the hospital; but before she could leave it, and while assisting in the wards, she was attacked suddenly with faintness and gasping for breath, and in a few minutes was dead. Her death took place seven days after admission.

The body was spare, and without œdema. The pericardium was healthy, the heart of moderate size, with parietes, lining membrane and valves all healthy. All the cavities contained dark fluid blood, without a trace of coagulum. The right lung was adherent to the parietes by an exceedingly delicate, easy-to-be-broken-down membrane. The organ was small, and its lower lobe partially collapsed. Its surface was studded with irregular, and in some cases, large patches of subserous hæmorrhage. Its tissue was crepitant throughout, though less so below than above. The bronchial tubes contained a large quantity of frothy mucus. The branches of the pulmonary artery distributed to the organ were in the greater part of their extent filled with decolourized and slightly adherent cylindrical coagula. They commenced, not in the trunk of the pulmonary artery, but in the division of it leading to this lung, formed casts of all the primary branches of this division, and were prolonged thence into many of the secondary and subsequent branches. The coagula, however, did not in all instances form parts of a continuous system; but in many cases the smaller branches, and in a few the larger branches, were occupied by coagula of the same kind as, but having no continuity with, those prolonged from the root of the lung. The clots were for the most part cylindrical, and accurate casts of the vessels in which they lay; still, here and there they presented slight irregularities and enlargements. They were for the most part adherent, though slightly so, to the parietes; but here and there were free, leaving passages between them and the arterial walls; which, together with the intervals entirely free from clot, were filled with dark-coloured fluid blood. All the clots presented a reticulated fibrinous surface, and a central black-currant-jelly-like axis. They had evidently formed prior to death. The pulmonary veins were empty. The left lung was in precisely the same condition as the right. There was no important disease in any other organ. The blood in the systemic arteries and veins was generally fluid; but in the left internal iliac vein, extending partly into the common iliac, was a cylindrical coagulum, not completely filling the vessel, but adherent to it, and presenting characters identical with those of the clot in the lung. Again, in the left innominate vein, a mass of coagulum was discovered, completely blocking it up; this was unadherent, and was found, on unravelling it, to consist of a branching system of partly decolourized clots, which could not have been formed there, but must have been carried thither from some of the smaller tributary vessels.

The above case, it may be added, is not adduced to prove that death may take place from the formation of clots within the pulmonary arteries. For the case is one in which there is room for difference of opinion in regard to the source of these clots. We believe, nevertheless, that in this case the clots found in the pulmonary arteries were formed in them; and we believe it partly in consequence of the form and structure of the clots, partly because there is no valid ground for disbelieving that such clots may be deposited during life, as well in the pulmonary arteries as in the systemic veins.

DISEASES OF THE PULMONARY ARTERY.

By R. Douglas Powell, M.D., F.R.C.P.

In any systematic consideration of the diseases affecting the pulmonary artery, that vessel must be separated into two portions, one external to, and one within the lungs. Disease of the pulmonary artery before its distribution in the lungs is so uncommon that, in practice, a murmur most audible over the region of this vessel is regarded as of hæmic origin, or this hypothesis failing, as attributable to some congenital defect about the heart, or to pressure from without, and it is only by this method of exclusion that we force ourselves to admit that the disease has its seat in the vessel itself.

Atheroma.

Ætiology.—The ætiology of atheroma and aneurism of the pulmonary artery does not essentially differ from that of corresponding affections of the aorta; but in consequence of the pulmonary vessel being more deeply seated, more lax in its capacity, and therefore less liable to direct injury or effective strain, the results of those constitutional influences, gout, alcoholism, syphilis, which lead to atheroma, are much more rarely developed. The milder degrees of atheroma—fatty degeneration of the intima—are, however, not unfrequently seen associated with those heart and lung diseases—mitral constriction and regurgitation, pulmonary fibrosis, or emphysema with hypertrophy of the right ventricle of the heart—which permanently increase the tension of the pulmonary circulation.

The association of such atheromatous patches in the pulmonary artery with those diseases which cause more or less persistent difficulty in the smaller circulation, and which have as their common accompaniment a more or less increased venosity of blood, is, as has been well pointed out by Drs. Wilks and Moxon, a strong argument against the supposition that this artery is protected from atheroma by virtue of the dark blood circulating through it. That the pulmonary artery is not wholly insusceptible to deeper lesions, however, is apparent from a case, to be presently cited, in which under the combined assaults of rheumatism, alcoholism, and hard work, with a very strong suspicion of syphilis, this vessel, in common the aorta, became affected with atheromatous disease resulting s of substance. Dr. Hope[1] refers to a case in which the vessel dilated and rigidly ossified, even beyond its primary divisions he lungs.

MPTOMS.—No symptoms have hitherto been traced as referable theroma of the pulmonary artery.

DILATATION. ANEURISM.

nder circumstances of great pressure within the pulmonary cir- ation, as in cases of marked narrowing of the mitral orifice, with t hypertrophy of the right heart, a certain general enlargement he pulmonary artery may take place. Dr. Sydney Coupland has rded the case of a naval pensioner, aged 75, in whom there was extreme degree of this general dilatation of the artery and its ches. In this case the main trunk was found dilated to a circum- nce of 6½ inches, the valves being incompetent and the walls of artery greatly thinned. There was in this case great hypertrophy dilatation of the right heart, marked emphysema of the lungs, and ly superficial atheroma of the intra-pulmonary branches. Although degree of patency of the foramen ovale was also present, Dr. Coup- regarded the emphysema as the real cause of the general dilata- f the vessel.[2] Dr. Conway Evans, in the Pathological Transactions 866, describes a case in which there was both general dilatation e artery and hypertrophic thickening of its walls, with atheroma e internal coat, associated with a contracted mitral orifice. Dr. s refers to other similar cases, recorded by Drs. Quain, Peacock, Bristowe, in all of which, as in the case he describes, marked rtrophy of the right ventricle was present.

case of aneurism of the main trunk of the pulmonary artery is ed to in Dr. Peacock's index to the Pathological Transactions, ols. xvi. to xxv. inclusive; and Mr. Erichsen, in his selected vations on aneurism, only refers to one case, described by Ambrose in which there was aneurismal dilatation and ossification of this l, from rupture of which the patient died suddenly. Dr. Crisp met with no recorded case.[3]

NARROWING OF THE PULMONARY ARTERY.

TIOLOGY.—Constriction of the pulmonary artery is most commonly ated at its commencement, and is generally a congenital disease, ciated with other congenital malformations of the heart, especially imperfect septum ventriculorum and patent foramen ovale and ductus Botalli (Lebert).[1] *Endocarditis* affecting the pulmonary valves and causing them to adhere by their margins, and so as partly to close the orifice, or *myocarditis*, leading to constriction at the conus arteriosus, are the most common causes of narrowing of the pulmonary artery: and these causes come into action before the end of the third month of intrauterine life.

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[1] Diseases of the Heart, 4th edit., p. 394, 1849.

[2] Path. Trans., vol, xxvi., 1875.

[3] Diseases of the Blood Vessels, 1847.

SYMPTOMS: DIAGNOSIS.—Dyspnœa and cyanosis becoming obvious as soon as the infant commences active movements, with the physical signs of great hypertrophy of the right side of the heart and a systolic murmur heard over the pulmonary cartilage and conducted upwards and to the left, are the principal points to be observed in the diagnosis of this malady. A more full consideration of its clinical and pathological features will be found in the section on congenital diseases of the heart.

Narrowing of the pulmonary artery beyond the valves is, like other diseases affecting this vessel, of rare occurrence. The most common cause of such local diminution of calibre is compression by a tumour, either aneurismal or from mediastinal growth, or possibly consisting of enlarged bronchial glands.

As the following case well illustrates the principal symptoms of compression of the pulmonary artery, and also presents other features of interest, I may perhaps be allowed to relate it here.

T. D., aged 38, a fireman in a pottery manufactory, came under my observation at the Charing Cross Hospital in October 1874. He was in his work exposed to great changes of temperature, sometimes working at a temperature of 200° or more, and also to pigment fumes and coal-dust, etc. To these causes he attributed a constant cough from which he had suffered for some time. He stated that he often had to lift heavy weights, as much as 2 cwt., which he would carry in his arms supported against the lower chest. No distinct history of syphilis was elicited; three children were living out of a family of nine. Patient had a severe attack of rheumatic fever in 1856 which lasted seventeen weeks, but had since then continued his work without difficulty up to six weeks previous to his attendance at the hospital. He had only for two weeks been quite disabled from work, on account of palpitation and breathlessness.

The following notes were taken a month before his admission into the hospital, where he continued under my care in the temporary absence of Dr. Silver. The patient was a short, stout man with a somewhat bloated face, of a dusky pallid hue, with decided lividity of lips. On exposing the chest an enlarged vein was observed coursing from the left shoulder along the second intercostal space to the sternum, the veins generally of the neck were full, and the carotids throbbed visibly. Pupils and pulses equal. The chest was expanded. The heart's apex beat at the sixth rib one inch outside nipple line, and, as indicated by shading in the diagram, the cardiac impulse was diffused from this point below the nipple to the epigastrium, and above the nipple to the second cartilage. Over a circular space B, having its centre at the third cartilage close to the sternum, and extending to the cartilage above and below, an impulse was felt synchronous with the apex-beat but more prolonged and attended by a marked thrill. Immediately succeeding this a second short impulse or shock coincided with the second sound of the heart. At the 2nd, mid-sternum, A, the systolic impulse was slight, without thrill, and the diastolic shock more faint. The superficial cardiac dulness was bounded, as indicated in the diagram, by a line extending from the second left cartilage near the sternum to the apex beat, and skirting the region of thrill and the left nipple. To the right the area of dulness reached halfway to the right nipple at the level of the fourth rib. A systolic rough bruit was heard most loudly over the region of thrill, B, and was succeeded by a second sound so accentuated as to communicate a shock to the ear. The blowing murmur was loud also at the base of the heart with marked accentuation of the second sound, and (?) a very slight diastolic bruit. Sibilant râles were heard over the chest; at the left posterior base there was some dulness extending round to the lower axilla with weakened respiration and subcrepitant râle. The systolic bruit was more audible at the left than the right suprascapular region.

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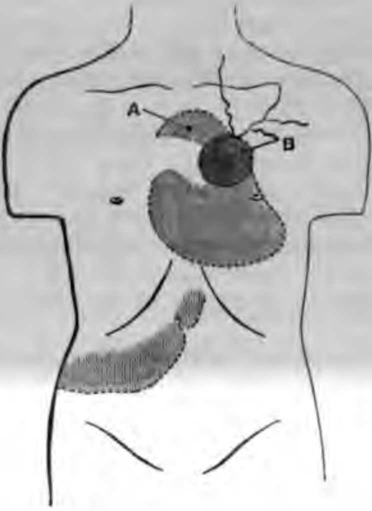
[1] *Vide* Clinical Lecture, Medical Times and Gazette, January, 1870.

The legs were œdematous, the liver enlarged, and the abdomen somewhat full, though not containing any considerable amount of fluid.

This patient continued under observation in the hospital until his death in December. Increasing dyspnœa with severe paroxysms, marked cyanosis, and extreme anasarca of the abdominal walls and lower extremities without much increase in the amount of fluid in the peritoneum, œdema of the lungs, increased congestion of the veins of the head and neck, with some regurgitation through the jugulars, were the principal signs of ingravescing disease. The physical signs about the heart did not materially change. The patient became intensely cyanotic shortly before death.

Bearing in mind the extreme rarity of aneurism affecting the main pulmonary artery, yet having regard to the signs of aneurism present, and to the evidence also, in the marked hypertrophy and dilatation of the right ventricle, of obstruction at the pulmonary artery, the diagnosis of aneurism of the aorta pressing upon and narrowing the pulmonary artery was inevitable. From the position of the dulness and thrill, and from the absence of any regurgitant murmur, it was assumed that the aneurism must be projecting forwards from the third portion of the arch. This, however, proved not to be the case.



At the autopsy an aneurismal pouch was found arising from the aorta a little above the valves, extending behind the pulmonary artery so as to project three-quarters of an inch beyond it, coming in contact with the left auricular appendix. This aneurism was found to have opened into the pulmonary artery. The left ventricle was greatly hypertrophied, the aorta atheromatous throughout, and from its inner surface several shallow pouches extended, each presenting a thickened margin.

The right ventricle was much dilated and hypertrophied, the pulmonary valves being natural. An oval well-defined aperture half an inch in diameter was found at the distance of three-quarters of an inch above the junction of the left and posterior valves. The vessel was somewhat stretched around the aperture and pouched inwards. Above the anterior valve at the same level was found a depressed smooth surface of irregular outline, having a raised, hard, puckered margin. The left ventricle was greatly hypertropied. The aorta was slightly contracted at its orifice, but immediately beyond the valves, which were healthy, it was dilated, and from the left side a wide-mouthed pouch extended behind the pulmonary artery, so as to project for three-quarters of an inch beyond it in contact with the left auricular appendix. Several shallow secondary pouches were to be seen on the inner surface of the sac, one projecting into the calibre of the pulmonary artery, and another having given way into it, forming the aperture above described. The liver showed lesions referable to drink and perhaps to syphilis.

This case is of pathological interest inasmuch as it is an example of a rare disease—atheromatous erosion of the pulmonary artery. Clinically, the symptoms and signs were principally those of stenosis of the pulmonary artery and aneurism of the aorta.

In this case the most important signs and symptoms were those most characteristic of constriction of the pulmonary artery beyond the valves. The systolic bruit most intense over the pulmonary cartilages, and here accompanied by systolic impulse and thrill with great accentuation of the second sound, were signs attributable to aortic aneurism ; but the marked evidence of hypertrophy and dilatation of the right heart, the general venous engorgement with regurgitation through the veins in the neck and general dropsy, pointed to an obstruction to the exit of blood from the right ventricle. In simpler cases the systolic murmur is characterised as pulmonary by its being conducted upwards and to the left, or to the left interscapular region. Accentuation of the second sound is insisted upon by Professor Quincke [1] as of importance in distinguishing constriction of the vessel beyond the valves from stenosis at or within the ventricular orifice when the second sound is obscured. There may be dilatation of the vessel above the point of constriction.

Dr. Peacock [2] in a tabulated collection of thirty-three cases of aortic aneurism opening into the heart or great vessel includes fifteen cases in which the communication was with the pulmonary artery. The aortic aneurisms in those cases arose with one exception from the ascending aorta, and in most instances immediately above the valves; the perforation of the pulmonary artery took place, with two exceptions, within an inch and a half of the valves. In two instances the vessel was perforated below its bifurcation.

These cases suffice to show that aortic aneurism is the most common cause of pressure upon the pulmonary artery. The degree of pressure from this cause must vary infinitely, and the cases must differ correspondingly in the relative intensity of the symptoms referable to the aneurism and to the compression of the pulmonary vessel. In the case above related the rupture of the aneurism only accelerated by a few hours the death of the patient, which was rapidly approaching, from symptoms referable to dilated right heart and obstructed venous circulation.

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[1] Ziemssen's Cyclopædia, vol. vi., Diseases of the Blood-vessels.

[2] Path. Trans., vol. xix., p. 126.

MURMUR OVER THE PULMONARY ARTERY.

A pulmonary murmur systolic in time, soft in quality, and of medium or low pitch, is commonly heard over the second and third left cartilages close to the sternum without there being reason to suspect any disease of the vessel. This murmur is very local,—not conducted in any direction. It is heard most frequently in young women and children, being then associated with arterial bruits in the neck, and with the venous hum audible over the jugulars (more especially over the right jugular, in which vessel a thrill may commonly be felt). Whatever the exact mechanism of the murmur may be anæmia is its most common cause. The pulmonary artery at its commencement is very superficial, and it is readily conceivable how the rush of a thin watery blood through an orifice so close under the ear should cause an appreciable sound. In children, and even in adults whose cartilages are tolerably resilient, the murmur may be produced or much intensified by pressure with the stethoscope; and it may be removed by such full inspiration as lifts the cartilages and ribs from pressing upon the vessel.

In extreme anæmia murmurs of the same kind may be heard all over the cardiac region, being generated at the several orifices of the heart.

In cases of retracted left lung from old-standing disease a murmur is frequently audible over the pulmonary artery, probably induced by the flattening of the chest wall bringing the cartilages in contact with the vessel. The tension of blood within the artery is always increased in these cases, as shown by the accentuation of the second sound, and sometimes in marked cases of fibrosis of the lung the division of the artery as it enters the affected organ is positively constricted and wrinkled. In other cases an enlarged and hardened gland will intrude upon the calibre of the vessel as it enters the lung. In displacement of heart from fluid effusions or other causes a systolic murmur may be heard over the pulmonary artery.

PULMONARY ARTERY WITHIN THE LUNG.

Although disease of the main trunk of the pulmonary artery is exceedingly rare, atheroma and even aneurism of its branches within the lung are frequently met with, but only, with equally rare exceptions, in cases of disease disorganising the lung, uncovering its vessels, and involving their walls in its destructive processes.

It is in the course of excavation of the lung in phthisis that thickenings, erosions, dilatations or actual aneurism of the branches of the pulmonary artery are most commonly met with. We have at once under these conditions the three most important determining causes of atheroma and aneurism, viz.: *increased blood pressure*, on account of the many vessels which are occluded, *local loss of support* from breaking down of the tissues around, and *softening of the arterial wall* by inflammatory changes, also quite of a local character.

In acute ulcerative destruction of the lung such vessels as do not become occluded in good time are apt to become softened or eroded, and, by their rupture, to give rise to copious and sometimes fatal hæmorrhage. In cases in which the destruction of the lung is less acute and violent, cavities form with more or less trabeculated walls, the trabeculæ consisting partly of bronchi, but chiefly of vessels surrounded by a certain thickness of condensed tissue. These vessels are as a rule occluded, but exceptions are occasionally met with. We frequently find in chronic cavities a large branch of the pulmonary artery which is quite patent, occupying a trabecula, or coursing along the wall of a cavity immediately beneath the limiting membrane. Such vessels, missing their wonted support on the cavity side, become strained by the blood-pressure. At first the arterial wall thickens,—not from hypertrophy, as Dr. Rasmussen has suggested,[1] for the thickening is limited to the side exposed, and has a uniform smooth section in which nothing but a commingling of connective tissue elements affecting the whole thickness of the wall and obscuring all distinctions between the coats can be seen. It is evidently an inflammatory process of the nature of endarteritis which affects these vessels, and although the thickening appears hard and fibroid, it nevertheless yields before the constant blood-pressure, the calibre of the vessel commencing to dilate at this point. Dilatation goes on in the usual way of aneurism, the originally thickened coat thinning as it becomes spread out before the increasing intrusion of blood, until it forms a brittle, soft, papery layer which cannot be recognised from a fibrinous lamina. Thus a most typical sacculated aneurism may form, projecting into a cavity more or less occupied by laminated fibrin, and which may rupture at any period of its formation.

The more chronic the cavity the more suitable the conditions for the formation of a sacculated aneurism.

In chronic cavities which have from exposure to some evil influences become inflamed or ulcerous, vessels imbedded in the walls, or occupying trabeculæ are rapidly laid bare, and may either become perforated or may dilate into irregularly shaped fusiform aneurisms. An attempt (often successful) at occluding such vessels may frequently be observed in the formation of a firm oat-shaped coagulum which is attached to the internal surface of the artery corresponding with the point of exposure. This plug may gradually enlarge and close the vessel.

There are some specimens in the Brompton Hospital Museum, showing sacculated aneurisms projecting into and occupying bronchial dilatations.

ÆTIOLOGY.—The ætiology of these aneurisms is included in their pathology. They are of strictly local origin, and it is doubtful if any constitutional conditions influence their production.

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[1] Edin. Med. Journ., paper translated by Dr. Moore, vol. xiv.

Age and Sex.—These conditions also only affect the occurrence of aneurism according as they influence the pulmonary disease. In fifteen cases which I have tabulated[1] the ages of the patients varied from fourteen to forty. I have since, however, met with a case of fatal hæmoptysis in an infant seven months old, from erosion of a dilated pulmonary vessel in a cavity.[2] Three of the above cases were females and the rest males: the infant just alluded to, however, was a female, and probably the preponderance of males in so small a number is of accidental occurrence.

SYMPTOMS AND SIGNS.—Copious hæmoptysis, repeated at short intervals, occurring in a case in which there is excavation of the lung, is the most characteristic symptom of the rupture of an aneurism, or a large vessel, within the lungs. The first gush of blood is of a dark venous colour but quite pure and unmixed. No other symptom or sign indicates the presence of these lesions of the pulmonary artery save in exceptional cases. In one case that fell under the notice of the author but a few hours before death, a very peculiar interrupted form of amphoric breathing was explained *post-mortem* by a small aneurism projecting into the chief bronchus at its point of communication with a large cavity.

DIAGNOSIS.—This can be made with tolerable certainty from the character of the hæmoptysis, as above explained. The more chronic and quiescent the cavity the more likely is sudden hæmoptysis to be derived from this source. Sometimes the excavation containing an aneurism is of very small dimensions.

PROGNOSIS.—Always of course very grave, but by no means necessarily fatal. Cases of the most profuse and oft-repeated hæmoptysis sometimes completely recover.

TREATMENT.—Absolute and prolonged rest with full doses of ergot give the best results. A very nice discrimination is needed in the management of these cases, especially as regards stimulants. It is at the moment of fainting that the best opportunity of coagulation occurs, and one must not be in a hurry to restore the force of the circulation. These patients, too, often recover and make blood very rapidly, and then are apt to get a return of their hæmoptysis. A restriction of diet, especially as regards butcher's meat, is often useful, and no stimulants should be allowed.

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[1] On the Pathology of Fatal Hæmoptysis. Path. Trans., vol. xxii., 1871.

[2] Ibid, vol. xxv. Dr. Fagge has since reported a case similar to aneurism occurring in a female child aged 2¾ years. Ibid. vol. xxviii.

ON DISEASES OF THE CORONARY ARTERIES.

By R. Douglas Powell, M.D., F.R.C.P.

The coronary arteries may be affected with any of those lesions—atheroma, fatty degeneration, calcification, occlusion, dilatation, or aneurism—to which other similar vessels are liable: and, as is also the case with like vessels going to important parts, the phenomena indicative of disease are all referable to damaged nutrition and disordered functions of the organ, in the present instance the heart, to which the vessels are distributed. The diagnosis of disease of the coronary vessels is therefore a pathological inference which is helped by no symptoms directly attributable to alteration in them. Extensive disease may exist without giving rise to any suspicious signs; indeed a moment's consideration of the conditions of the coronary circulation suffices to enable us to see that they may be varied or interfered with independently of disease affecting the vessels themselves.

The coronary arteries are, unlike other systemic vessels, filled at the moment of cardiac relaxation by the systole of the aorta forcing the blood back upon the closed aortic valves. If the aortic valves be damaged so as to admit of free regurgitation, the pressure of blood in the coronary arteries is thereby more or less diminished, and the vigour of the circulation through them lessened. Again, atheromatous disease of the aorta at its origin not infrequently leads to almost complete closure of the coronary vessels at their commencement. Other morbid conditions, such as an undue rigidity of the aorta or aneurism affecting it may by interfering with the rebound or systole of the vessel materially influence the coronary circulation. Hence those disease-phenomena, such as angina, or irregular or failing heart's action with syncopal attacks which, when no more definite physical signs are present, are regarded as being due to fatty degeneration of the heart in consequence of a diseased condition of its vessels, may equally be due to disorder or derangement of the circulation through the vessels arising from some one of several other causes.

Atheroma, calcification.—*Angina pectoris* is frequently connected with calcification of the coronary arteries, but by no means necessarily so. Of three of the most rapidly fatal cases of angina ever recorded, viz. those related by Dr. Latham,[1] in only one was disease of these vessels present to any appreciable extent. Dr. Dickinson, in the seventeenth volume of the Pathological Transactions, calls attention to occlusion of the coronary arteries at their commencement as a cause of angina. He relates three cases in which "soft atheroma spreading under the lining of the aorta" had caused great narrowing or complete closure of the mouths of the vessels which were otherwise quite healthy. The muscular substance of the heart was in each case slightly fatty but not atrophied. A similar case is recorded by Mr. Spenser Watson in vol. xix. of the same Society's Transactions. Professor Gardiner has more fully treated of this subject in the Fourth Volume of this *System of Medicine*. Dr. Quain[1] has shown in how large a proportion of cases of true fatty degeneration of the heart the faulty nutrition is traceable to diseased vessels.

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[1] Diseases of the Heart, New. Syd. Soc. Edit., p. 450, *et seq.*

THROMBOSIS.—Dr. Hayden[2] refers to the occasional occurrence of thrombosis affecting the coronary vessels as a cause of acute fatty degeneration of the heart. In a case of Dr. Quain's,[3] in which the aorta was dilated, the left coronary artery was found to be completely obliterated at the first part of its course and occupied for an inch further by an adherent clot, apparently the result of thrombosis. In this case great cardiac agony was experienced by the patient, only relieved by sedatives for two months before death; there was however only a slight amount of fatty degeneration present.

ANEURISM.—Aneurism of the coronary artery has been met with as a "museum curiosity" in several instances. Dr. Gee records a remarkable case in the St. Bartholomew's Hospital Reports, vol. vii., in which three aneurisms were found upon these arteries in a boy aged seven years, who had died with scarlatinal dropsy, pneumonia and meningitis. In the St. Thomas's Museum Catalogue[4] a specimen is described showing aneurismal dilatations along the course of the coronary arteries varying in size from that of a pea downwards, sacculated, some empty, others completely filled by adherent buff-coloured clot. The heart was removed from a man aged 22, who had died of pulmonary apoplexy and hæmorrhage into the kidneys, and who therefore was presumably the subject of general arterial disease.

The materials are not at present available for any further clinical consideration of diseases of the coronary arteries. In minute anatomy these diseases present no peculiarities.

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[1] Med. Chir. Trans., vol. xxxiii.

[2] Diseases of the Heart and Aorta, p. 1017.

[3] Path. Trans. vol. xxiii. p. 57.

[4] Vol. iii. No. 81.

INFLAMMATION OF THE LYMPHATIC VESSELS.

By J. Russell Reynolds, M.D., F.R.S.

Affections of the system of lymphatic vessels are closely associated with diseases of the skin, of the glandular apparatus, and of other organs which may be the seats of dyscrasic and diathetic disease; and hence the major part of their pathology as well as of their clinical history will be found in the articles on Erysipelas, Hodgkin's Disease, Leucocythæmia, and Pyæmia. Sometimes, however, from so-called "accidental" conditions, an inflammatory process may occur, independently of any one of those more general changes in the organism; and this may exist in such form in the lymphatic vessels as to merit a separate notice.

Synonyms.—The terms Adenitis, Angeioleucitis, Lymphangitis, and Lymphadenitis, have been used to denote this state.

Causes.—These may be placed in two categories: (1) those which are simply accidental, such as exposure to cold, wounds, bruises, strains; and (2) those which carry with them some toxic agent which affects the body generally, and, it may be, mainly through the lymphatic vessels. When the lymphatic vessels are the seat of inflammatory change, as the result of "accidental" injury, it is to be found that the constitutional state is unsatisfactory. There is to be traced some taint, either hereditary or acquired since birth, which disposes the individual to lymphatic disease, and without which a mere bruise or wound would have been inoperative. Occasionally rapid inflammation has occurred after a simple injury; when some poison has been introduced into the body and has become the starting-point of Lymphangitis, the body may have been previously healthy, but the impression so made upon it may be such as to lead to most mischievous results.

The most common causes are injury to the nails, especially of the foot, chronic ulcers of the skin, stings, punctured wounds, bites, the introduction of unwholesome animal matter from wounds, abraded mucous surfaces, morbid mucous secretions, or any tissue undergoing unhealthy change. The surface of the wound may absorb some poison from the air; diphtheria has been followed by this disease; the vessels in proximity to cancerous, tubercular, or other morbid growths, may become the seat of inflammation.

SYMPTOMS.—The classical signs of inflammation are those which constitute the local indications of Lymphangitis, viz., pain, tenderness, redness, and swelling. These are obvious when the superficial vessels are inflamed, but less distinct when the more deeply seated lymphatics are especially involved. The pain is not, as a rule, severe; there may be only stiffness, or a stinging and burning sensation. The tenderness is in proportion to the superficiality of the inflammation and its association with dermatitis, either simple or specific, and it is sometimes very great. The redness, sometimes of vinous hue, is observed to run in long narrow lines along the course of the vessels, and often forming a network extending from the periphery towards the trunk, and reaching laterally beyond the lymphatics. The swelling of the vessels may be distinct; they are hardened, knotted, and enlarged, but the changes they exhibit do not pass downwards. The glands into which the lymphatics pass become speedily inflamed, and the skin and cellular tissue are involved in a general inflammatory process, usually of erysipelatous sort. The inflammation of the vessels usually stops at the gland nearest to the seat of injury, and œdema of the skin and subcutaneous areolar tissue exists beyond the site of inflammation.

The course of the disease may be rapid; and, when associated with some classes of poisons, rapidly fatal, passing into suppuration, sloughs, or gangrene; but, on the other hand, the inflammation may be resolved; or it may pass into a chronic state, with much induration of skin, and hypertrophy of some of its elements.

The general symptoms, like those of erysipelas, vary with the nature of their cause. They may be slight when secondary to a merely local injury; but severe and of adynamic character when the result of poisonous infection.

Usually there is a feeling of chilliness, rather than a rigor, at the onset, followed by irregular alternations of heat and cold, with trembling of the limbs. The pulse is always frequent, but variable in force and volume. There is nausea and præcordial discomfort, followed by vomiting, insomnia, and delirium. Such febrile symptoms may precede the appearance of local changes, and become aggravated as the latter are developed. Rigors, attended with profuse sweating, and acccompanied by distension of the abdomen, dyspnœa, very frequent pulse, and muttering delirium, are the signs of approaching death by blood-poisoning.

DIAGNOSIS.—Phlebitis may resemble Lymphangitis in its mode of origin and in many of its symptoms, but differs at its commencement in the more distinctly localised character of the ailment, in the larger size and smaller number of the red lines which mark its existence, in their greater hardness, and less frequent tendency to become associated with changes in the cellular tissue. The swelling is less, the pain not so severe, and the general disturbance less pronounced. It must be remembered, however, that the two conditions may co-exist.

From erysipelas this disease may be distinguished by the presence of those special vascular changes which are observed in inflammation of the lymphatics, and which are not present in erysipelas. The latter affection is of relatively shorter duration, exhibits more general inflammation of the skin, and is frequently associated with general toxæmia, much more highly marked than in Lymphangitis.

It is sufficient to say that Lymphangitis has been sometimes mistaken for simple erythema, or erythema nodosum, to put the practitioner on his guard against a repetition of such errors.

STRUCTURAL CHANGES.—Thickening of the walls of the vessels, infiltration of the connective tissue in their neighbourhood, pus in and about them, glandular suppurations, and sloughs, are the most common appearances. The skin is often covered with phlyctenæ, or with spots of gangrene, while the central organs may present no departure from health, or only such as are common to all toxæmic and adynamic states. Secondary abscesses are sometimes found in liver or in lung, and phlebitis is by no means uncommon.

THE GENERAL TREATMENT of Lymphangitis requires no special notice, as it differs in no respect from that which is required for the various maladies of which it forms a part; and the local treatment of its complications is such as falls into the province of the surgeon, and requires no description here.

§ V. DISEASES OF THE BLOOD-GLANDULAR SYSTEM.

A. THE SPLEEN.

- INFLAMMATION.
- HYPERTROPHY.
- NEURALGIA.
- TUMOUR.
- SOFTENING.
- INDURATION.
- ATROPHY.
- HÆMORRHAGE AND RUPTURE.
- GANGRENE.

B. LEUCOCYTHÆMIA.

C. THE LYMPHATIC GLANDS.—HODGKIN'S DISEASE.

D. THE SUPRA-RENAL CAPSULES.—ADDISON'S DISEASE.

E. THE THYROID BODY.—EXOPHTHALMIC GOITRE.

DISEASES OF THE SPLEEN.

By John Richard Wardell, M.D., F.R.C.P.

Diseases of the Spleen most frequently prevail in marshy districts and tropical countries, and as the complication of intermittent and remittent fever. Acute Splenitis implies inflammation of the serous investment, which sometimes extends from the parenchyma, but which, in the majority of instances, occurs by continuity, when other internal parts are inflamed. Inflammation of the Spleen, when not referrible to wounds or external violence, is never a primary but always a secondary complaint. The most common affection of this organ is that of sub-acute or congestive splenitis. The viscus may be very large and turgid without being inflamed, and such state sometimes comes on with much suddenness. The parenchyma is far less prone to acute inflammation than the envelopes. The organ may be the seat of great pain, constituting splenalgia, or dolor lateris, when quite uninflamed. It passes into suppuration, and abscesses of various sizes are formed in its structure. It is often greatly enlarged in European children who are resident in the tropics. In leucocythæmia the organ attains very great magnitude. In dyscrasia, and especially in that degradation of the circulatory fluids which obtains in periodic, continued, malignant, and exanthematous fevers, the Spleen undergoes marked appearances of organic change. Morbid anatomy discovers, as in the other viscera, the ordinary inflammatory products, hypertrophy, atrophy, induration, softening, and gangrene. On its tunics are sometimes witnessed fibrinous, cartilaginous, and ossific conversions. In its substance are occasionally seen fatty degeneration, amyloid deposition, hydatids, cysts, and the tuberculous, cancerous, osseous, and calcareous changes.

General Terminology.—Σπλήν (Gr.); Splen (Lat.); Rate (Fr.); Milz (Ger.); Splenitis (various authors); Lienis inflammatio (Boerhaave and Van Swieten); Splenitis phlegmonodea (Sauvages and De Haen); Splenalgia suppuratoria (Sauvages); Empresma splenitis (Good); Splenitis peritonealis acuta (Craigie); Splenite (Fr.); Entzündung der Milz (Ger.). The non-inflammatory affections have been variously named Congestion, Turgescence, and Hyperæmia of the Spleen. And when pain has been the chief characteristic, Splenalgia, Dolor lateris, Splenis Dolor, and Splenic Pain, are terms which hav been commonly employed. In certain districts it is usual to speak o its chronic enlargement in the vernacular, as Disease of the Milt, o the Ague Cake.

There is scarcely any part of the human body of which more has been written and respecting which more theories have been propounded than the Spleen. Its doubtful purpose in the economy has evidently constituted the main cause of the great consideration which has been bestowed upon it, and of the numerous and varied hypotheses which from distant ages have been enunciated. The most ancient account which we have of this viscus and the diseases to which it is liable is to be found in those writings which are attributed to Hippocrates, and where the organ is so prominently mentioned in relation to the well-known doctrine of the four humours. When Haller began to write on this subject, he premised his observations by saying that he was plunging into the region of mere conjecture, darker than in the case of any other viscus. Though much respecting its use has in recent times been made out, still there remains, as relates more especially to its functions, a good deal that is speculative and not fully accepted, and in the commencement of this article the writer cannot feel unimpressed with the figurative but emphatic declaration of Haller. The knowledge which we have of its office, and the pathological changes to which it is subject, is by no means so clear and absolute as our knowledge of most of the other internal parts. It would be out of place here to give in any lengthened detail the doctrines which have from time to time been put forth and successively fallen into oblivion, but to some of these a passing reference may be made. In remote ages it was remarked that an enlargement of the Spleen was generally accompanied by an emaciated condition of the body. In the Hippocratic collection, it says "those persons whose Spleen is large have their body meagre." The same idea is to be found in the *Timæus* of Plato, and which, it is related, gave rise to the well-known comparison of Trajan, who said that the Imperial treasury was like the Spleen, because when that was rich the people were impoverished. Aristotle believed the Spleen to assist the liver in digestion, that it attracts from the stomach superfluous and excrementitious humours (*ἰχμάδας τὰς περισσάς*), and corrects them. Plato supposed that its office is to relieve the liver when distended. Galen, whose opinions were evidently more widely accepted, and longer assented to than any of the opinions which before his time had been advanced, imagined that the humour called black-bile (*χολὴ μέλαινα*) is secreted by the Spleen, in the same way as the yellow bile (*χολή ξανθή*) is secreted by the liver. The Arabian physicians and the few philosophers, who, in the sparsely scattered seats of learning, lived in the dark ages, were evidently contented with the Galenic theory. Aretæus had arrived at the idea that the Spleen is nourished by black-blood (*μέλαινα χολή*), and hence the terms *melancholy* and *splenetic*. Orbasius, Trallanus, Paulus Ægineta, Actuarius, Haly

Abbas, Protospatharius, and Meletius held to the views of Galen. Erasistratus deemed it as being of no real use in the body. Avicenna conceived that, being an organ so surcharged with blood, warmth is imparted to the stomach, and thus the process of digestion becomes aided; and certain of the Arabic writers entertained the chimerical notion that its office is to cool and refresh the heart. Such were the crude conjectures and vaguely defined doctrines which have been transmitted from ancient times. They were the mere expressions of speculation and the flights of fancy, without being based upon inductive reasoning, and having little or no foundation in experiment. With certain modifications they were held until the beginning of the sixteenth century, that epoch which is regarded as the period of the revival of letters and philosophy, when the works of the ancients became more carefully and more generally studied, and their opinions were subjected to closer examination and nicer criticism. Some of the anatomists who then flourished looked upon the vasa brevia as ducts passing from the stomach. Franciscus Ulmus imagined the Spleen to be possessed of certain properties necessary for the preparation of the blood for the heart and arteries, and Tel Plater also was of opinion that it had the function of elaborating the blood, rather than the functions of attracting the melancholic humour (Gray.) Other of those remote writers, as St. Ambrose, modified the Galenic notions and contended that the Spleen draws away the useless part of the aliment, and after its retention for a time transfers the purified and subtle remainder through the liver to the blood. Less antiquated hypotheses than those of the Grecian and Roman philosophers are to be found in the works of the Schoolmen who flourished in mediæval times; but they likewise lay claim to no serious regard, as their theories, like the theories of those who had long preceded them, were but the chimeras of caprice and delusive imaginings, devoid of proof, and readily to be contradicted by experiment. Some thought the organ secreted a fluid which passed into the stomach; some, that it elaborated a product necessary to the nervous system, and others that it produced a sort of chyle.

Coming down to more modern times, and even to recent periods when physiological problems have been cautiously tried by, and only found an accepted solution in accurate and repeated experiments, still the entire uses of the Spleen have not been finally determined, and many distinguished names have been the adherents of opposing views. Müller thought its importance in the economy less than many other authors supposed, and averred it fallacious to maintain that it is essentially connected with the function of the liver. Mead and Meyer deemed it necessary to digestion, Tiedemann and Gmelin were convinced that it generates a certain kind of lymph which serves to form the blood-globules, and Kölliker, Funke and Billroth incline to the last-named supposition; Dollinger regarded it as a sort of symmetrical appendage to the liver; Lieutaud and Moreschi looked upon it as a reservoir to accommodate the blood of the stomach when that organ becomes distended with food, and when more blood is sen to it during the process of digestion; Sir Everard Home revived the Hippocratic theory of the Spleen receiving fluid from the cardiac end of the stomach to be thence carried into the circulation; and Ecker and Beclard say that one of its chief uses is for the solution and destruction of the blood-globules. Microscopical anatomists, and especially those of Germany, first paved the way to more precise and definite conclusions relative to this organ, and of late years pathologists have been induced to study its varied diseases with more interest than was formerly the case. If reference be made to systematic writers on this subject, it will be seen that there is not that satisfaction, not that conviction, expressed which is to be observed in the articles treating on the ailments incident to the other viscera. The difficulties which have beset authors in their descriptions of the Spleen and its maladies have arisen from a variety of causes, and not only from a want of knowledge of its functions, and amongst the chief of which causes may be instanced its peculiar situation in the abdomen, being deep in the left hypochondrium and surrounded by organs which are regarded as more prone to disease, and with whose symptomatology physicians have been long more familiar; by its being hidden as it were by the thoracic wall; by its capability of sudden and even great distension without giving rise to any serious inconvenience in the system; by its comparatively simple action; by its low degree of sensibility; by its diseases not being endemic in this as they are in malarious countries; and by there being an erroneous impression that splenic complaints are by no means common. From what, therefore, has now been said there are difficulties in the diagnosis of its lesions which do not obtain with many other organs. Being a ductless gland, and not influencing any secretion or excretion, nor giving off any secreted product that can be submitted to examination and experiment, its morbid phenomena cannot be studied and comprehended with the same certitude and exactness with which we can investigate the diseases of the liver and the kidneys. The vascular glands, of which the spleen may be regarded as the prototype, though they differ from the secernent glands, are importantly connected with the process of sanguification. By some modified mode of secretion they abstract certain materials from the blood, act upon such materials, and then restore them to the circulation without being eliminated by any duct or outlet from the organ. There is then great analogy in function between the blood or ductless glands and the secreting glands. Between nutrition and secretion there is great analogy, and the processes proper to vascular glands are partly nutritive and partly secretive. That other organs can vicariously assume its office in the economy, and that it is not absolutely essential to health or even to life, is evident because it has been extirpated in the human subject and the patient has lived thirteen-and-a-half-years and enjoyed health. It has been found so exceedingly small as to be quite in a rudimentary state, and it s been said to be altogether wanting. Even in health, as Bright serves, its volume, consistence and position may vary from so many ses that it is not subject to the same rules and precision in the vestigation of its maladies as can be brought to bear upon der viscera. The advances which microscopical anatomy, experimental physiology, and animal chemistry have latterly made, and are ll making, promise much in clearing up certain mysteries which t remain, as I have already remarked, with regard to the Spleen. It is becoming more admitted, and it is certainly incontestable, that en in the British Isles, where the endemic element in the causation of its morbid affections obtains to an infinitely less extent than in former times, it is more frequently the seat of diseased action han the older authors were led to suppose. The increased tention which clinical teachers have recently given to physical gnosis in all forms of abdominal complaints has contributed more and will contribute still more to the comparative accuracy with which we can judge of its lesions.

It may here be remarked to the reader that a knowledge of its position in the abdomen, of its anatomical and microscopic structure, and, so far as we can speak, of its functions, is essential to a right comprehension of its ailments. Hence, in attempting to treat of this subject in any systematic work on medicine, such must needs be done in a somewhat exceptional manner. It must be remembered that in health the Spleen cannot be felt. If it can be felt and is not really diseased, it is then displaced. Lying obliquely in the left hypochondrium and in intimate relation with the diaphragm, by which it is separated from the ninth, tenth, and eleventh ribs, its situation necessarily varies with the act of respiration—a fact never to be forgotten in diagnosis. In the deepest expiration its upper end will ascend to the lower edge of the eighth rib; and in the deepest inspiration, when the organ is normal in volume, it never descends below the cartilaginous margin of the chest. It is attached to the stomach and the pancreas, and at its lower angle it touches the left kidney. Its size varies more than that of any other organ in the body, except the uterus and the ovaries. The greatest weight of anatomical evidence goes to prove that this viscus is a single organ in the body. In its earliest development it appears in the median line. Dr. Embleton thus expresses himself on this point: "The Spleen originates in the median line of the body, is gradually carried thence to the left hypochondrium, and it ought to be regarded as originally median and not belonging to the left side more than the right." This writer then cites a number of ancient and modern authorities who look upon it as a single organ.

The *peculiar structure* of the Spleen is not only a circumstance carrying with it much physiological interest, but it has a direct and an important pathological significance. Its distensile qualities intimately concern its lesions; indeed this property constitutes one of the main causes of its most frequent morbid changes. Its capsule is of fibrous structure and invested by the peritoneum, and this tunica propria, being of elastic tissue, enables it to yield to the greater or less distension of its vessels. It everywhere encloses the parenchyma. From the inner surface of the elastic tunic sheaths are formed for the vessels and their ramifications, and from these sheaths fibrous septa and bands (trabeculæ) are given off innumerably, and these form cells (loculi) into which the blood is effused. This fibrous coat is composed of white and yellow fibres, the first named consisting of parallel bands and the last mentioned being united in a densely reticulated arrangement. According to Kölliker, the trabecular tissue, in its elementary essentials, closely corresponds with the fibrous capsule, since it exhibits both white and yellow fibres; and he considers them as being muscular, thus on this point agreeing with Malpighi and others who had persistently regarded the partitions of the Spleen as being muscular. One of the most recent writers (Marshall) on this particular question, entertaining the same opinion, says: "The proper coat, the sheaths of the vessels, and the trabeculæ consist of white fibrous and areolar tissues, mixed with elastic fibres, and contain, especially in animals, pale fusiform, unstriped muscular fibre-cells." The splenic capsule is very yielding, and the walls of its vessels are unusually thin. This slight elasticity accounts for the slow disappearance of the organ's distension when from any cause it has become surcharged with blood. The mode in which infected blood still further diminishes its distensile properties is mainly, as it would seem from the experiments of Jaschkowitz, by a greater or less degree of paralysis induced in the branches of the sympathetic which go to this viscus. There is no doubt, however, that mere and continued mechanical pressure exerts its effects upon these nerves.

There is no organ which receives a greater number of blood-vessels in proportion to its size than the Spleen, and its extreme vascularity has always been dwelt upon by anatomists as one of its distinguishing features: and this fact, as Bichat remarks, becomes more worthy of notice because it secretes no fluid. The splenic artery, which is the largest branch of the cœliac axis, and which near the organ breaks up dichotomously into five or six branches named the *rami splenici*, is a very large blood-vessel in proportion to the organ it supplies. When these rami enter at the hilus, they divide into innumerable subdivisions, nor do they anastomose, and they soon divide into a coarse capillary network, and this retiform structure quickly ends in minute veins. The splenic vein is of very considerable calibre as compared with the artery and the volume of the viscus. According to Home, Piesker, and others, it is five or six times larger than the artery, and its coats are very thick and it has no valves. It constitutes the way of return, by many tributaries, for a large volume of blood, as it takes the venous blood from the pancreas, duodenum, the greater part of the stomach, and sometimes the left colon and part of the rectum.

The red splenic pulp or parenchyma occupies the spaces between the trabeculæ and exterior of the vessels. It is a soft, bluish-brownish mass, which becomes redder when exposed to the atmospheric air. It consists of a colourless, granular parenchyma, mixed with coloured cells and red blood-corpuscles, the colourless portions being composed of variously shaped nucleated cells and a finely granular plasma, and which, it has been said, resembles the ultimate materials entering into the formation of the solitary and agminated glands of the intestines. Physiologists regard the coloured cells as altered red blood-corpuscles and as being peculiar to the spleen. Some are smaller than the ordinary blood-globules, others are yellow, brown, or black, and their pigments may be seen in depositions which are crystalline or granular. In the spleen-pulp are the Malpighian corpuscles, which are small white, soft bodies, measuring from the sixth to the third of a line in diameter; they are attached to the small arteries and trabeculæ, and arranged in sessile manner like buds on a twig, but they have no communication with the arteries, and their contents are granular and nucleated cell elements. They are so numerous that they are said to form one-fifth or one-sixth of the splenic pulp. It has been noticed that they are absent in the human Spleen after protracted disease and in starved animals, but very observable in health and when the body had been highly nourished.

The nerves of the Spleen are derived from the solar plexus, and they form an interlacement called the splenic plexus. The nerves are relatively small as the vessels are relatively large, a fact acknowledged from the time of Aretæus, and hence the little sensibility with which this internal part is endowed. The nervous filaments follow the course of, and may be traced in, the minute arterial branches which go to the Malpighian corpuscles, and pass into the pulp. Embleton believes the gland may receive nerve-twigs from each pneumogastric and the sympathetic ganglia on each side; such anatomical arrangement has not yet, however, been fully determined.

During the process of digestion the spleen is temporarily enlarged, which can be understood when it is considered that the vasa brevia are branches of the splenic artery which inosculate with the gastric artery and the gastro-epiploica sinistra. When the stomach is distended with food, more blood is sent to those vascular connections, and the Spleen, by its accommodating structure, receives any undue supply of blood which, during the activity of the digestive functions, is determined to these parts. When there is loss in the force of cardiac action, its congestion is a consequence; and when the volume of the circulation is diminished by hæmorrhage, or when irritation in the gastro-intestinal membrane is followed by serous diarrhœa, it can at once be comprehended how this viscus will be reduced in size. Physiological experiments have gone to confirm the opinion long ago advanced by Hewson that this gland is engaged in the formation of the germs of the blood-corpuscles. It is probable, though not absolutely certain, that it is also engaged in their disintegration. M. Picard has recently demonstrated that the organ contains a proportion of iron four times greater than that of the blood itself. It is by some confidently stated that in diseases of this organ, pigment masses, the débris of broken-up blood-cells, have been discovered. The blood-cells of this organ are of varied colour, and it is probable they are not only there disintegrated, but it has been conjectured that materials from the red corpuscles, such as the pigments, may be again used in the germs which go to constitute the new corpuscles. Kölliker not only regards its functions as concerned in the renewal of the blood, but he believes, with the ancients, in the secretion of bile also. This doctrine is, however, a mere hypothesis. That the Spleen acts as a diverticulum to the visceral circulation is much more certain than anything we know of its other uses, and that it is destined to a more exalted office than that of being a sort of mechanical contrivance, or vascular reservoir, is now sufficiently conclusive, for had such been its mere purpose, there would then, as it has been remarked, have been no need for its peculiar parenchyma, and a simple plexus of blood-vessels would have subserved such end. Towards the termination of the digestive act, not only is its entire volume increased but there is also an absolute augmentation of the spleen pulp, and also of the Malpighian bodies. It attains its largest dimensions at the termination of chymification, and if no food for some time be taken, it then becomes gradually reduced in volume. The resilience of its elastic tissues renders it particularly adapted for taking in a surcharge of fluid during the torpor of the vascular system which results from digestion. In disease its peculiar capabilities are made subservient to the altered requirements of the circulation. In hepatic affections, more particularly in cirrhosis, it can be comprehended, from the foregoing physiological facts, how frequently the Spleen is involved. The blood of the splenic veins must pass through the liver, and if there be obstruction in the last-named organ, a backward retardation in the circulation of the Spleen must necessarily be produced. In chronic cardiac disease, and especially when the right side of the heart is enlarged, this organ from venous obstruction becomes tumid. In children it should be borne in mind that the Spleen, like the liver, is relatively large. Gray considers its office is to balance the quantity as well as the quality of the blood, which it is able to do by its connection with that part of the vascular system which is concerned in introducing new materials into the circulation. Its contraction and relaxation, it is possible, may be influenced by the sympathetic system, because electrical currents, as it has been shown by experiment, cause the organ to contract. The views of Carpenter relative to the use of the Spleen seem so apt that they may here not inappropriately be quoted. "We are inclined to believe," says this eminent authority in a review, "that the office of the *colourless* parenchyma of the Spleen is not only to serve as a storehouse for the surplus albumen that finds its way into the circulation on the completion of the digestive process, but also to excite an *assimilating* action upon it, whereby it is rendered more fit for the nutrition of the tissues, and of this assimilating action we deem the generation of fibrin to be one of mals. And if it be true, as we have elsewhere suggested, that pecial function of the red corpuscles is to assimilate or prepare peculiar combination of materials which is required for the tion of the nervo-muscular apparatus, the disintegration of these cles in the splenic parenchyma may answer the twofold purpose gulating their total proportion in the mass of the blood, and of ng, through the liquor sanguinis, the materials which the nerves scular tissues are to draw from it for their own development."[1] rani, from experiments on the lower animals, concluded that pleen has the double function of presiding over a metamorphosis nic elements and of accumulating iron for hæmatin. And kowitz found that section of its nerves caused to it increased f blood and increase of hæmatin-pigment. If we acknowledge s being certain of the functions of the Spleen, much light is necessarily thrown upon those affections with which its more manifest es are associated.

relation which subsists between the Spleen and the lymphatic s, which was first pointed out by Hodgkin, and the discovery excess of white corpuscles in the blood in the form of its ic disease, are facts which have added very much to our knowledge of its functions, and will doubtless be followed by still more e information. It has also been stated that there is some n between the Spleen and Peyer's patches, and it is a point of erable interest, as Sir William Jenner observes, that the Spleen, mphatic glands, and Peyer's patches all suffer involution at the period of life—about fifty. This writer also remarks that at ime the Spleen becomes less, the lymphatic glands waste, and 's patches smooth down; and that it is at this period of life that seases, and especially enteric fever, in which these parts are ed, cease to be common.

ERAL ETIOLOGY.—Diseases of the Spleen are limited to no ular period of life; we see them in infancy, in middle age, n advanced years; and males, from their more frequent out-r occupations and consequently greater exposure to external nces and their less temperate habits, are more liable to them emales. With respect to the causes, none is more prolific than hich is endemic; and to malarial agency, splenic lesion, and was maintained. The physicians in mediæval times concurred in these views. Modern authorities, more especially those who have written on the diseases of tropical countries, and such as are met with in the vast range of our colonial dependencies, have abundantly pointed out the malarial origin of the majority of the ailments incidental to this viscus. In the fenny counties of England, more especially those of Cambridge, Lincoln, and Essex, from time immemorial to that in which we live, splenic complaints have prevailed. In the East and West Indies; in the Southern States of America, and particularly in the extensive river plains of that continent; in China, Canada, and in Australia; in Hungary, in Holland, in Italy, and in those countries bordering on the shores of the Mediterranean, this etiological influence is so well known as to need not more than a passing reference. In tropical countries, where great heat and moisture prevail, or those parts formed by the deltas of great rivers where the soil is composed of alluvial and vegetable remains, and where paludal exhalations are given off, the causation is rife. Inundations from the sea, and great inland floods, or where sea and fresh-water for a time stagnate and then disappear, render low grounds extremely insalubrious, and there the affections considered are in great degree produced. In Holland, where these inundations have so often occurred, the effects described have as the rule always followed. In that country, after the partial inundations by the sea in some places in the winter of 1825 and 1826, during the following summer, when the sun's rays dried up the earth which had been so long saturated, the decomposition of organic matters ensued, and the concentrated effluvia were succeeded by fevers of a remittent type with splenic complications. From the accounts which have been given of the endemic diseases in Hungary, in the summer and autumn, when the hot weather has promoted the decomposition of organic materials, fevers of an adynamic type with Spleen affection very commonly prevail. In former times, when in our own country vast tracts of forest and marsh land lay in a state of nature, when the kingdom was thinly populated, and before reclamation and drainage became so general, the maladies incident to this organ were often observed. Again, paradoxical as it may appear, cultivation and drainage may have a deleterious effect. It has been repeatedly affirmed that in the tropics and in our colonies, where the ground was thick with trees and overgrown with rank vegetation, that such places were more healthy than immediately subsequent to the efforts of the cultivator. The trees and undergrowth keep off the sun's rays, but when they are removed, and the soil becomes first subjected to heat, miasms are then far more abundantly exhaled, and it requires a succession of seasons—sometimes many years—before the new ground has fully given off the poisonous gases which under such circumstances had been generated. Dr. Rush has recorded the kind of effects now spoken of which took place after the clearings in the primæval forests of Pennsylvania, and in the works of Montfalcon and Bailly similar

ny is given. Sometimes higher situations are more dangerous
in than the situations beneath them, because the miasms may
ed away by prevalent winds, and the emanations may be con-
ed in the vapours which rise from the valleys, and carried
is to be intercepted by acclivities and neighbouring eminences.
ct has been long observed, and the explanation now advanced
en generally acknowledged. The various records of our military
tions, and the statements made by medical men who have
d in our colonies, abundantly attest the correctness of these
s. These authorities also have told us that the new residents
h places are far more prone to malarious attacks than those
ave in some measure become acclimatised. Great and prolonged
lar exertions, which not only determine the blood to the
al organs, but absolutely produce changes in the constitution
e fluid, such as protracted and fatiguing marches and the
us operations of active service, together with great privations,
ed, the use of stagnant water and water which has run through
y districts, sleeping in damp situations or in the open air
ut sufficient covering, and mental depression produce splenic
laints. Blane and Dawson, in their respective accounts of the
eren diseases, give prominence to such causes. Sudden transi-
of temperature which occur in those climates where hot days
cceeded by cold nights, and where periodic rains produce a
fall in the thermometer, have been mentioned by various writers.
lood is then quickly determined from the surface to the visceral
, and from what has already been said of the accommodative
istensile qualities of the Spleen, it can be comprehended how
abrupt repletion of its vessels must, if such surcharges were
nously repeated, tend to the permanent enlargement and struc-
alteration of the viscus. Periodic fevers thus produce its
rophy. Sir J. Ranald Martin thus expresses himself on the
now considered: "The most ordinary cause of splenic con-
n," says this writer, "whether active or passive, will always be
in the malarious countries of the East—remittent and inter-
t—which, for longer or shorter periods, and by the recurrence of
old or congestive stages for months or years together, disturb or
ally destroy the balance of the abdominal circulation, and with
integrity of the abdominal functions. When to these morbid
ions we add destitution, the absence of comfort in food and
g, the residence in low, cold, and damp localities, mental
sion, those causes, in short, which contaminate the blood, and
ine its flow into the abdominal organs: all these causes will
ally tend to the production of splenic disease."[1]
bid changes in the blood itself, effected in a more direct and in
nediate manner, are to be noticed; as the absorption of noxious
, which at once operate upon the circulatory functions and are
d by visceral turgescence; the specific poisons of the various

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[1] "Tropical Climates," second edition, p. 501–2.

forms of fever, and the retention of effete matters from the imperfect action of the depurating organs and especially of the kidneys. In those contaminations of the blood associated with pyæmia, septicæmia, or icorrhæmia, the Spleen not infrequently assumes the purulent condition. There is no doubt that as the common event this gland becomes tumid where there is metastatic infarction and when infectious thrombi circulate in the blood. The loss of nervous power consequent upon some depressive agency affecting the great nervous centres which diminish vital cohesion, and those cachexial changes which proclaim debility, are also to be enumerated.

Twining speaks of an assemblage of symptoms, all caused by debilitating influences and tending to asthenia, which obtain notably in Bengal, affecting not only the European residents but also the native population, and which he calls the endemic cachexia of the tropics, as being a condition markedly associated with splenic complaints. In the complications and sequelæ of intermittent fever, this organ is very commonly attacked. Pemberton says it is most frequently affected by quartans. In the continued fevers, more especially in those instances in which there are hepatic symptoms and where the adynamic type is assumed, its disease is very commonly noticed. Tweedie speaks of often having found it soft and enlarged. Barrullian in 1,202 cases of typhus discerned the Spleen diseased in 126 instances, or in 10·48 per cent. Louis states that in forty-six inspections of enteric fever, in only four cases was it healthy. Murchison also gives testimony of its frequent affection in this form of fever. Birch-Hirschfeld says that in enteric fever Spleen tumour is generally most marked in the second or third week. In relapsing fever Cormack says it was congestively enlarged in a great number of cases. Warburton Begbie, in his description of this fever, asserts that the organ now spoken of is almost always altered. In an account of relapsing fever which I published in 1847 I then said the Spleen was well-nigh always diseased. In the Russian epidemic of relapsing fever in 1864-65, as described by Tillner, Doubowitski, and Bernstein, it was large. Wunderlich and Steffen thus also speak of it. In the exanthems it is not uncommonly found morbid. In the epidemic of cerebro-spinal-meningitis, which occurred in Leipsic 1864-65, and was recorded by Wunderlich, the Spleen was found large, with a tendency to hæmorrhage in such cases as were distinguished by great exhaustion, palpitation and vomiting.

That diseases of the liver are often followed by lesions of this organ is an acknowledged fact. The splenic vein is one of the great formative trunks of the portal vein, and thus when the circulation of the liver is impeded, there is very commonly, but not uniformly, congestion of the Spleen. Murchison says this result is less frequent than might be imagined; and when the circulation of the liver is mechanically obstructed, he believes the Spleen is rendered large in about one-half of the cases. He believes that the absence of enlargement is consequent on the fibrous thickening or calcification of the capsule, ating vascular dilatation, or on an excessive drain from the o-intestinal mucous membrane by diarrhœa or hæmorrhage. portal system being destitute of valves, the splenic vein has con- atly communication with the whole of the portal system. In tic disease, and especially when the action of the liver becomes bled, it can readily be comprehended how in a conservative manner lood can be diverted into the spleen. Of cirrhosis splenic disease e frequent accompaniment. An undue afflux of blood is sent to retained in this multilocular reservoir, and such surcharge of is succeeded by its enlargement. Rheumatic endocarditis and nic diseases of the heart, especially of the mitral and tricuspid es, and of the great vessels, are also to be instanced. According rch-Hirschfeld of Dresden,[1] there may be considerable enlargement uncompensated mitral insufficiency, but as the rule there is no ble splenic increase when there is merely incompetency of the c valves. When in acute rheumatism fibrinous depositions have me attached to the valvular margins, and are subsequently dis- grated and carried into the current of the circulation, these broken-pieces of fibrine are sometimes, in the Spleen as in other organs, ted in the minute vessels, and by giving rise to capillary em- m set up inflammation. It is now known as one of the advances odern pathology, though perhaps of less frequent occurrence, that n the edges of the cardiac valves have been rendered rough by the ologous transformations of age, and when deposits of fibrine from blood-stream have taken place upon the margins of the valves, fibrinous pieces may be washed off, and by being transferred to Spleen, as they are transferred to the brain, the liver, and the eys, and perhaps to other viscera, may produce capillary embo- precisely resembling that which occurs in rheumatic carditis. this order of sequences is doubtless an etiological explanation of e inflammatory patches and purulent collections of smaller or r extent which were by Louis, Abercrombie, Craigie, and others idered as examples of idiopathic inflammation, and of those con- lations which Rokitansky referred to the doctrine of crasis. hysema of the lungs, by producing dilatation of the right cavities he heart, and consequently hepatic engorgement; interruption to flow of the catamenia; the suppression of hæmorrhoids and of neous eruptions, have long been assigned as causes. Bree gives cause depressed circulation from external cold, and he also says Spleen may be idiopathically affected. Idiopathic splenitis is, ever, now a term inadmissible, as its inflammation, when not ting from mechanical injury, embolism, or from continuity of ture, or the proximity of other inflamed organs and parts, is ys preceded by lesion of the circulating fluids. External injuries alls, blows, or compression may be instanced. Long walks, drink- cold drinks when the body is much heated, sudden chills after piration and fatigue, are also causes which may be named.

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[1] Berliner Klinische Wochenschrift, Nov. 30, 1874.

GENERAL SYMPTOMATOLOGY.—When this organ becomes morbidly affected in its acute, sub-acute, and congestive forms, the predominating indications are a feeling of pain or tenderness, or a sense of weight or oppression in the left hypochondrium, left shoulder, and left side; the breathing is often short or shallow; the patient cannot lie easily on the right side, and not uncommonly complaint is made of fulness and tension, which extend from the left side into the stomach. There may be sympathetic pains which continue through into the back and up into the left shoulder and the back of the neck. Dr. Embleton has published an able paper,[1] illustrated by several cases relative to shoulder-tip pain in splenic diseases. He believes, as before observed, that the splenic nerves are derived from each side of the semilunar ganglion and from each of the members of the par-vagum, and thus by receiving nerve-twigs from each pneumogastric, he says the shoulder-pain of the right side may be accounted for. Grotanelli pointed out the frequent occurrence of pain in the left scapula and shoulder as an indication; Cruveilhier gives much significance to shoulder-pain as a symptom; and Bigsby, Bree, and Copland insist upon the same fact. Of ten cases of splenitis given by Embleton, in six the left pneumogastric was found tender on pressure, the right nerve in two, and both nerves in one example; and this physician says pains in the left loin, left leg, left ear, temple, and left side of the head, from the nervous connections above mentioned, may be symptomatic of splenic disease. And he thus continues: "This shoulder-pain in splenic disease will limit on the left side most commonly, as it does generally in hepatic diseases on the right side, the respiratory movements of the sterno-cleido-mastoid and trapezius muscles, and thus the splenic side of the chest will be kept more quiet than it would otherwise be. When liver and Spleen are both diseased, then both sides may be similarly affected in their movements. The above morbid state of the pneumogastric trunk may and does in its upward course affect various branches, as those of the stomach, liver, heart, lungs, and even ear, giving origin to symptoms characteristic of disorder of one or other, or of more than one of those organs; and an attentive observer may remark that many of the symptoms in cases of Spleen disease are attributable to parts which receive branches from the pneumogastric nerve." These facts are of much importance in judging of the symptoms of subjective character. The condition of the countenance is very indicative of splenic disease, and to the practised eye of those who have seen much of the affection in malarious countries, its presence is recognised almost at a glance. There is a heavy, dull, listless, apathetic expression which betokens that lethargic state of the mind with which the ailment is so commonly accompanied. The face is often of a dirtyish lemon colour, sometimes it looks as if puffed and swollen; the tongue, which is generally more or less coated with a whitish fur, is pale, and sometimes as if transparent; the soft parts of the throat are bloodless and flaccid, and the lips are blanched; the conjunctivæ are of bluish white, and the general facial indication is emphatically anæmic. In the acute stage, and more especially when we have reason to believe the serous covering to be inflamed, the pulse is quick and full, and in some cases it is somewhat incompressible. In the sub-acute, congestive, and more chronic forms of its lesion, the digestive or assimilating process becomes impaired, there is unusual epigastric fulness, the bowels are confined, the stools are mostly dark and may contain blood, and the urine is high coloured. Sometimes diarrhœa sets in, and there may be hæmorrhoidal loss of blood or an increase of uterine discharge. Thirst, anorexia, and elevation of temperature in greater or less degree obtain. In a week or ten or twelve days there is generally some decline, in the acuter forms, of the more urgent symptoms, and the disease becomes gradually resolved, or some sequential event, as suppuration, ensues, when other and more characteristic phenomena are presented. In the sub-acute, congestive, or latent examples of its lesion, which are far more frequently witnessed, as in those in which its affection is consecutive or the complication of another malady, as in adynamic, simple, or remittent fevers, the symptoms are to some extent—occasionally a good deal—masked by the primary complaints, and it becomes difficult to decide as to how much of the general disturbance and the more objective symptoms depends upon the specific fever and how much depends upon the disease of this organ. In the chronic forms it has from the earliest times been remarked that there is often a tendency to hæmorrhage both from trivial causes and spontaneously, that ulcerated surfaces do not heal as under ordinary circumstances, and that the cheeks and gums sometimes become gangrenous. In females in malarious countries the uterine functions become much disordered in splenic diseases.

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[1] Brit. Med. Journ. Sep. 19, 1874.

In reviewing the symptoms of splenic disease, it is all-important to bear in mind what has already been said relative to the position of this organ in the abdomen; and there are conditions and circumstances which, if overlooked, would vitiate our conclusions and give rise to erroneous diagnosis. In deformed thorax, as in rachitic deformity, this organ may be moved from its proper place. When the diaphragm is more concave than natural, the spleen ascends abnormally. In lung emphysema; where tumours are formed in the substance of the diaphragm; in effusion into the left chest, and in encephaloid infiltration into the lower part of the left pulmonary substance, it may be felt at the costal edge when not diseased. It does not normally lie so high up under the chest-wall in children as it does in adults, because in children the diaphragm is not so concave. It may be held as the common rule that when the Spleen can be touched it is diseased. Palpable tumour is the surest sign of its lesion, and our decision should be arrived at from negative as well as positive facts. The neighbouring organs should be severally excluded. When the stomach is large and resonant, and when the descending colon and the left arch of the colon are distended with flatus, the examination of the viscus will be more difficult. When the organ is considerably enlarged, palpation and percussion discover diminished resilience. It is then brought nearer to the lower chest-wall and to the abdominal parietes. The practised hand will readily perceive the difference which is given to the fingers, and the dull, dead sound which percussion elicits will at once be recognized. Sometimes when moderately enlarged it is borne obliquely forwards and inwards, and being carried from the chest-wall, percussion discovers little or no increase of the normal area of dulness. When acutely affected, as it often is in a secondary manner in specific fevers and in pyæmia, digital examination readily produces pain; in its chronic enlargement palpation gives little or no pain. When the increase is considerable, its mechanical pressure, as I have seen, by impinging upon the walls of the stomach, is apt to give rise to the frequent feeling of nausea and sometimes to sickness and vomiting. It may from foregoing inflammation of the diaphragmatic covering and adjacent parts of the peritonæum become adherent, and when it is thus adherent to the diaphragm it may not descend with the inspirations. From its loose attachments, its smaller size, and its greater motion during respiration, it is less liable to adhesion than the liver. It may also be morbidly united to the stomach when its up-and-down motion is rendered limited. Some pathologists affirm that it is very rarely adherent to the diaphragm. It is sometimes thus attached in chronic tubercular peritonitis.

When the diaphragm on the left side is acutely inflamed, as it sometimes is in pleuro-pneumonia, or when it is the seat of malignant deposit, its impaired functions prevent the rising and falling of the viscus, and its motion may be rendered hardly perceptible or be absolutely abolished. In empyema, when the diaphragm becomes stretched and paralysed, the same result is observed, and the organ may be thrust down below the costal edge, and thus morbid enlargement be simulated. When its enlargement is considerable it pushes up the diaphragm, forms a mechanical impediment to the expansion of the left lung, interferes with the action of the heart, and dyspnœa, coughing, and palpitation may become urgent symptoms. When the above-named conditions occur, the physical examination of the thorax will throw much light upon the splenic symptoms. It must be remembered that in some cases of its enlargement it may extend quite down to the pelvis and beyond the linea alba. In leucocythæmia it may attain these dimensions. The feeling which when enlarged it confers to the fingers is that of smoothness and resistance, and of being immediately under the abdominal parietes. Its notched and sharp edge, when felt, leaves no doubt as to its identity. When chronically enlarged the patient's gait acquires a peculiarity; he involuntarily inclines to the left side; he relaxes the abdominal muscles on that side, and accommodates himself to the uneasy feeling which he experiences; and it has been remarked that there is more freedom of motion in the right than in the left extremity. When the disease has long continued there often is a deathly pallor of the countenance, tinged with a greenish hue, and which is peculiar to the diseases of this organ. Sir William Jenner thus speaks relative to the symptoms of splenic affections:—"A moderately enlarged Spleen forms a tumour in the abdomen situate on the left side and passing upwards under the margin of the thorax. Even when of moderate size, the tumour lies near the surface; and there is no intestine in the front of it. In these particulars it differs from the kidney. It has a sharp anterior margin, and in this also it differs from the kidney. The anterior margin of the tumour passes from above downwards and inwards. Sometimes, but only infrequently, one or more notches can be felt in this border; when felt the notch is quite characteristic. The lower border of the splenic tumour is rounded; the posterior border is to the left, but not so distinctly as the anterior and lower borders. The fingers can, however, always be passed in behind a tumour formed by an enlarged Spleen. You see the resistance of the Spleen as you pass your hand back and back till you reach the space just outside the mass of muscles that lie in the spinal groove, and then you find a narrow space where there is very little resistance; the anterior border of this space is formed by the posterior border of the Spleen. Having one hand on the posterior border of the tumour, and the other in the front on the anterior edge of the tumour, you find that it is movable from side to side. You can push the anterior border towards or away from the middle line; you move the Spleen thus when it is enlarged, because it is very rarely indeed closely adherent. You know how loosely the Spleen is attached by the mesentery, and it is infinitely rare for the inflammation of the capsule of the Spleen to be followed by adhesions sufficiently close and numerous to destroy its mobility."[1]

When the left lobe of the liver is enlarged—and it may be enlarged much more than the right, but in nearly all instances where one lobe is diseased the other is diseased also—it may be mistaken for the Spleen. Bright observes that in such cases the margin of the liver may be traced running towards the right side. The enlarged Spleen, it should be remembered, lies anterior to the liver. A deep inspiration will generally solve the difficulty, because the Spleen more distinctly rises and falls than the liver, and it descends lower than the left lobe into the abdomen. In hepatic lesion the common symptoms of jaundice, dulness, and tenderness in the right hypochondrium, and the colour of the alvine and renal excretions, will assist in conducting to a right conclusion. Left kidney enlargement may simulate enlargement of the Spleen, and more especially when the former is encysted. The left kidney is placed nearer the spine, it is more fixed, the intestine lies before it, and if the patient is requested to place himself on his hands and knees, the kidney does not drop forward so much as in this position the Spleen will drop forward. In renal tumour, the anterior upper and under borders are smooth;

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[1] Brit. Med. Journ. Jan. 16, 1869.

there is no sharp defined edge to be felt as there is in the spleen and left lobe of the liver. Coughing and deep inspiration do not move the kidney, as they move the Spleen, downwards. In cystic formations and turgid growths of the kidney the tumour can be traced forwards and towards the umbilicus, and if traced backwards the dulness is continuous and uninterrupted towards the spine. The kidney has its seat close to the lumbar muscles; there is no space between it, as there is space between the Spleen and these muscles. In doubtful cases, as to whether the enlargement be renal or splenic, the ordinary tests and the microscopical examination of the urine will aid in making a correct decision. In renal disease the pain often takes the course of the ureter, and in the male there is mostly pain in the direction of the testicle. Renal abscess may resemble enlarged Spleen, but renal abscess extends back more completely into the lumbar region; it is fixed, and does not alter its position when the patient is turned on the right side, and it feels tense and elastic to the fingers. Bright remarks that an accumulation of fæces sometimes renders the diagnosis extremely difficult. But Jenner says fæcal tumours are very rare. They may be most certainly judged of by their altering their position; their configuration, as ascertained by digital examination, varies; sulcated irregularities and depressions can be felt; and the adjacent colon is often tympanitic. There is also often the history of constipation, and of constipation alternating with liquid stools. A dose of castor oil or a large enema will in nearly all cases remove any doubt entertained. Ovarian tumour will be recognized by the ordinary symptoms of that form of disease. In such cases the tumour is first felt lower down in the abdomen, and its enlargement is more rapid. Percussion elicits resonance between the costal edge and the lower defined dulness. When the fundus of the stomach is the seat of cancer, and a large cancerous mass is formed in that situation, the general cachexia, the sickness and vomiting, and the loss of flesh will commonly indicate the malignant complaint. In scirrhus of the omentum the tumour is rough, hard and knotted; it stretches like a flattened indurated mass over the front of the abdominal viscera; it is not confined to one side; it is not traceable up under the costal edge like splenic tumour, and pain and sickness I have known to be most distressing symptoms in cancer of the omentum. Subcutaneous collections of purulent matter over the regions of this organ have been said to resemble splenic enlargement, but their superficial and stationary positions render their true nature distinguishable. Acephalo-cyst hydatids may rise from the spleen. Such cases are exceedingly exceptional, and when they do occur their elastic, rounded, smooth, bossy form will make them known when carefully examined.

GENERAL PATHOLOGY.—Organic lesions of the Spleen are, in the great majority of instances, secondary or sequential. They arise from some form of inflammation, in many cases as the result of ary or obstructive causes. There may have been less of vital ion in the gland or certain morbid alterations in the circulating a. From the relaxing and yielding tissues of the Spleen abnormal es of blood in it is much favoured, and when such surcharges n been long continued and frequently, repeated interstitial changes pt to follow. Perversion in the function of the organic nervous em tends to such fluxions in this viscus. Experiment has shown division of the branches of the sympathetic which go to the organ eeded by its turgescence. Dorsdoff and Botschetschkaroff of St. burg made a series of experiments relative to the swelling and action of this viscus. They observed all its diameters enlarged n the nerves of the splenic plexus were divided, and that these eters were diminished when the peripheral ends of these es were electrically excited; they noticed the liver to become ented in volume, denser and of lighter colour when the spleen as artificially contracted, and that with each of its contractions e blood-cells were discharged into the hepatic blood. It would m, too, from the researches of these experimenters, that the contion and enlargement of the organ are, not as Müller and others maintained, wholly dependent upon vaso-motor function, but upon the influence of its muscular elements.[1] Such being the effect tting off the normal nervous influence, it can be understood when lood is infected by malarial and specific poisons how contractility mes diminished and varying morbid processes are instituted. When is obstruction and closure of the portal vein, as sometimes takes in hepatic disease, such as in cirrhosis and cancer, or when there umours which produce mechanical pressure, congestion of the en may occur in marked degree. In heart and lung disease its gement is less common. The parenchyma is less prone to inflamon than the envelopes of the organ; and this fact can readily be prehended when the structure of the respective tissues is coned. From the statements of Gray and Kölliker the parenchyma is tituted of microscopic fibrous filaments, capillary-vessels, pulp-and blood-globules, in various states of dissolution, the pulp-cells bling the Malpighian corpuscules, only being smaller. The pighian corpuscles are imbedded in the pulp, and always attached minute branch of an arterial twig, united to its extremity, resting a vessel or situated at the angle of division, and are filled with d albuminous substance which contains nucleated cells. Such being minute arrangement of its structure, a considerable and sudden t of blood may be accommodated. The first condition of its most mon morbid state is that of congestion. Its proper vessels become rged, the trabeculæ lose their elasticity, the capsule and its s covering are unduly stretched, and the inflammatory process rvenes. Pemberton vaguely attempted to account for this liarity by saying there is a want of proneness in the arteries to on, as he termed it, the essence of inflammation. In tissues more dense and resisting, the irritation which vascular pressure confers sooner and more demonstratively proclaims the inflammatory phenomena. Bree thinks pyrexia is prevented by the turgid and accommodating qualities of the vessels. In the process of inflammation, its substance is apt to be broken up and the pulp rendered semi-diffluent, being mixed with shreds of trabeculæ and vascular sheaths. The antecedent morbid changes of these more manifest conditions are an occult impairment of the organ's vital endowments, whereby follow excessive interstitial exudation of plasma, which is unsuited to healthy nutrition; the blood-globules become more than normally dissolved, and the fibrine is increased; and perverted nervous action so influences vascular action as to affect the splenic cells and capillaries. There is, doubtless, between the intertrabecular spaces and the vessels some kind of communication not yet understood. The blood constantly passing from these alveolar cavities carries with it cellular elements; but if from any cause the onward flow becomes impeded, there must be morbid increase of the pulp, because the cells are then abnormally accumulated. In certain acute diffusive processes the loss of the natural blood stasis is very apparent. According to Billroth, the ultimate and molecular changes of the Spleen in typhus are very characteristic. In the veins he found a surprising quantity of cells containing from two to six nuclei, the normal cells being greatly diminished. In this disease the Malpighian corpuscules exhibit a decrease of cell formation. The large cells in typhus Spleens diminish relatively with the duration of the fever. In pyæmia and septicæmia the morphological change most usual is tumefaction of the cell elements. In hæmorrhagic infarction the venous sinuses are generally discovered in a thrombose condition, the blood-globules being matted and massed together in heaps. In putrid and adynamic fevers it sometimes appears as if the pulp had been abolished and the body of the organ converted into a dark grumous fluid, the coats being loose and flaccid, easily torn, and the contents being readily poured out. Softening and enlargement of the viscus are the constant conditions in malignant fevers. In periodic fevers this soft and deliquescent process not unfrequently goes on into the suppurative state. Pus is then found in a number of small deposits, or in a single large collection. The existence of suppuration cannot, however, always be determined upon, and it has been present when physicians of great practical acumen have not even suspected it. According to Andral, pus in minute yellow drops sometimes infiltrates the entire substance of the viscus. It is generally of the creamy kind, but Cruveilhier says it is occasionally concrete. When the suppurative result has taken place, the membranes wellnigh always become affected, just as in hepatic abscess the peritoneal tunic becomes inflamed and dissection discovers lymphic adhesions. The organ may thus be agglutinated to the stomach or the colon, ulceration may take place, and the purulent secretion be poured into one of these hollow viscera and be vomited or passed by the bowel.

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[1] Med. Times and Gaz., Vol. I, 1876, p. 308.

Under the continuous and great tension of the capsule, and when it has been rendered tender by disease, it may give way and the pus be extravasated into the cavity of the peritoneum, when diffuse and flagrant peritonitis is speedily established and death soon follows. Sometimes in acute splenitis adhesion takes place to adjacent parts with little injury or inconvenience.

In more exceptional cases, when the process of inflammation has been protracted, there is an indurated condition not only of the envelopes and the fibrous tissues of the organ, but, also of its parenchymatous substance. This state is mainly caused by the condensation of the coagulated matter contained in the cells. There is abnormal deposition of the lymph, and in such examples the cartilaginous tendency is exemplified in the denser structures. There is hyperplasia of the interstitial connective tissue.

Chronic is of far more frequent occurrence than acute inflammation of this organ, and it is this form which is so frequently connected with intermittent and remittent and also other forms of fever. It is sometimes the consequence of hepatitis, dysentery, chronic diseases of the liver, and more especially, as before remarked, of obstructed portal circulation, and organic affections of the heart and large vessels, and when the natural crasis of the blood has been impaired. In malarious countries it is a malady very frequently presented to the physician, and it is from the accounts of those who have resided in the tropics, and whose deductions have been made from a large amount of experience, that we have the best and amplest descriptions. The chronic may be the result of the sthenically acute form, but such examples are exceptional, as slow and gradual congestion is the common forerunner of the kind of lesion now more particularly considered. The earlier stage of the complaint is often masked by the ordinary symptoms incident to the intermittent or remittent attacks, and in proportion as these diseases are in their course marked by asthenia, the splenic complication is less prominent and less easy of recognition. It has sometimes, when the physical signs have been neglected, made an insidious advancement before detection, and sometimes its lesion has not been discovered before dissection. It may be large, and its lesion obtains when there are no subjective signs. The patient tells you he can lie on either side, takes a long breath without uneasiness, and complains of no dulness or aching under the left chest. The older physicians, who knew nothing of the kind of physical examination now practised, had to depend upon more general symptoms, and consequently they not unfrequently altogether overlooked this viscus. Bree says it may have made great progress before being observed, and that there may be pain with little enlargement, and that there may be great enlargement with little pain, and that the pulse may give no evidence of the malady. Pemberton states that it may be thus diseased without producing any uneasiness whatever. William Hunter, De Haen, Grottanelli, and various Indian authors and more recent writers expatiate on the covert nature of the affection and lay emphasis upon its insidious character. Being frequently the accompaniment or sequel of intermittent or remittent fever, the severity or mildness of its lesion varies much according to the nature of the endemic influences, the preceding health, and the vital powers of the individual. Enlargement is the most notable of all conditions of chronic splenitis; but this should not be the only object of inquiry, as such condition is but the manifest state of a great and foregoing malady pervading the entire system, and we should give due attention to those antecedent and existent changes by which it is produced. An extraneous and deleterious agent is imbibed into the body, and the entirety of the circulating fluids undergoes a potent and morbid alteration. It may be that the nervous centres are primarily affected, and more especially the nerves of organic life, and that the organ which, as it has already been said, is so importantly, so eminently concerned in the evolution of corpuscular germs of the blood, may in greater degree than in any other organ be impressed by the poison. Other poisons, which give rise to specific fevers, exemplify their peculiarities in affecting certain and distinct organs and tissues; and the cause of their diversity of action can only be in certain subtle and essential differences in the primary and extrinsic agent. Accompanying the chronic ailments now considered are great debility, languid circulation, deficiency of red-blood globules, and a yellowish, pallid unhealthiness of aspect. The expression is dull, the mind apathetic; in addition to the dirtyish lemon hue of the skin, the facial integument is sometimes puffed and bloated, the eye looks clear and blanched, the lips, tongue and fauces are typical of anæmia. Muscular debility and general inertness are apparent, and in this lowered condition the functions of respiration, assimilation and secretion become gravely affected. That hæmorrhage, dropsy, or gangrene, should sometimes follow the train of morbid processes now instanced cannot be surprising. Such depravation of the fluids is inevitably succeeded by more obvious and pronounced pathological changes. Cullen had long ago pointed out how the kind of splenic affections now more particularly referred to may end in resolution, suppuration, or gangrene. It has been stated above that chronic splenitis may come on without subjective symptoms, and it sometimes happens that it may make very slow progress, and with little or no suffering. In other instances there is some discomfort from tumid belly and difficult respiration; and there may be alternative periods of improvement and remissions of an aguish character for many months, or it may be for years.

In certain examples of excessive splenic hyperæmia, and when the fluxionary surcharge of the organ has been sudden, the symptoms of anæmia quickly supervene, and more speedily than could be looked for in any general blood change. The temperature may, as it frequently does, rise high during the intermittent paroxysm; but it may be equally high in continued fever, and the tissue oxidation and blood consumption be as great; but the symptoms of anæmia are far more emphasised when the periodic type has preceded. Mechanical causes doubtless explain this peculiarity. The symptoms of bloodlessness are produced relatively in rapidity and degree with the suddenness and extent of the splenic enlargement. Such a congested condition of this organ means really the abrupt and temporary withdrawal of a large quantity of the vital fluid from the system. If the hyperæmia quickly decrease, the anæmic symptoms also begin to disappear, and with more quickness than would be the case if such depended on the improvement of impoverished blood. A general amelioration in the circulating fluids would require far more time. The experiments of Griesinger have shown the correlation which subsists between these symptoms and the rapid enlargement of the viscus. The fluxionary hyperæmia incident to the respective types of continued fever is neither so excessive, nor does it produce the kind of symptoms now particularly considered. When the forms of continued fever have run their course, and convalescence succeeds, the organ is restored to its normal condition. In remittents it is not so, and more especially if the paroxysms have been often repeated. Continued tension effects alteration in fibroid and vascular structures, and augmentation of volume follows.

The morbid condition of the blood which is now known as melanæmia is doubtless referable to an abnormal process carried on in this viscus; for nearly all observers now regard the spleen as the place where the pigment is formed in the melanæmic dyscrasia under the influence of the malarial cachexia. The German pathologists were the first to investigate this subject, and prominently point out the manner in which the pigment is formed in pernicious intermittent fever. According to Rindfleisch, pigmentary particles are discovered floating in the blood. Their shape is irregular; they may be yellow or brown, but are more commonly black granules; as the rule, they are smaller than the blood-corpuscles, but a few may be seen which are of larger size. Some are spindle-shaped corpuscles, others are round, and the particles of free pigment vary in their physical configuration. Trousseau, in speaking of this pigment, says it is mainly formed of granules which are amorphous, sometimes irregularly agglomerated, and sometimes cylindrical, from having been moulded in the interior of vessels. The product now spoken of is consequent on the transformation of hæmatine. The tradition that black substances are generated in the spleen and blood is one of the oldest doctrines in the history of medicine. From the days of Galen to the times of Van Helmont, Sylvius, Boerhaave and Van Swieten, the cause of this condition was under various theories discussed. Certain of the ancient writers were of opinion that the fluidity of the blood under particular and specially perverted processes in the organism was diminished, and that a black and condensed product resulted; and some enunciated the notion that the more solid character of the new substance gave rise to mechanical obstruction in the viscera, and more especially in the brain, and thus were accounted for those grave effects of which it was the forerunner. It was the doctrine of Hippocratic humoralism still maintained, but under particular modifications. Kämpf added to atrabiliary matters other substances produced in the plasma of the blood. Reil in the last century opposed the ancient notion of the physiological effect of black bile, and subsequently Heusinger and Puchelt attributed the pathological phenomena to the deposition of a black pigment. Lancisi and Stohl had long before noticed a dark tinging of the skin and black deposits in the brain of those who had died of malarious fever; and, coming down to later times, Bailly, Bright, Annesley, Haspel, and Stewardson commented upon this morbid condition. In 1837, Meckel fully and satisfactorily ascertained that this dark colouring of organs and tissues depended upon pigmentation derived from the blood, and he was of opinion that the pigment formed in this gland passes into the portal blood, that the larger particles are detained in the hepatic lobular zones, and that the finer particles pass through the liver to be transferred to various parts of the body. The blockage which it effects has been called microscopic embolism. Virchow, Heschl and Planer more recently gave illustrative examples of this now commonly received fact. The spleen is the organ where the pigment is manufactured, and more especially when that organ has been repeatedly and excessively congested. In the stasis of hyperæmia the blood-globules are decomposed and their hematine becomes transformed into pigment. It is then carried into the vena porta, into the liver, the vena cava, and into the heart, thence to be diffused into the general circulation. Some American observers have known pigmentation confined to the portal blood. The malarial poison causes splenic hyperæmia, and consequently extensive destruction of red-blood corpuscles. Frerichs says, in the hyperæmia of the spleen, consequent upon intermittent fever, the stagnation is exceedingly great, and as a result the formation of large masses of pigment. This writer also believes that, in exceptional cases, the liver, and not the Spleen, may possess the capability of making pigment. He gives a case in which none was discovered in the spleen, but in which much was detected in the liver. As the melanæmic dyscrasia is not always observed in splenic enlargement consequent on pernicious intermittents, and as occasionally pigment is present when the hyperæmia is not excessive, a mere mechanical way of accounting for this result can hardly be admitted. It is exceedingly presumptive that the marsh miasm under certain conditions and in certain epidemics causes excessive necrosis of red-blood corpuscles. The relative virulency of poisons will determine the degree of necrotic power, but of the altered physical conditions giving rise to such effects we have no means of judging. The experiments of Virchow lead him to believe that in great destruction of the blood-cells in the spleen the hematine passes into the colourless elements of the spleen-pulp and by this means enters the blood. This writer is also of opinion that the bright substance adhering to the pigment granules consists of the protein matter which was combined in the fibrin of the disintegrated corpuscles. Rindfleisch says pigmentation occurs in the intervascular cords of the spleen-pulp where the blood flows most slowly; and as the intervascular cords are not shut off from the cavernous veins by an impermeable membrane, filtration goes on between the arterioles and the venous radicles, and in this way pigment flakes pass into the blood. In those organs in which the capillaries are the narrowest pigment deposits are most marked. It is thus that black matter is so frequently discovered in the brain. The retia mirabilia of the kidneys are unfavourable to the passage of these particles, and thus the kidneys next to the brain are most liable to melanæmic pigmentation. Such being the obstructive qualities of this product, the pathogenic theory has been promulgated that the graver symptoms of pernicious fevers may be attributed to pigmentary embolia. Extensive destruction of blood-globules from the cause now described must necessarily be followed by a general impoverishment of the blood, and thus in marsh fevers, anæmia; hence that peculiar colour of the face in malarial cachexia, and which is so characteristic of chronic disease of the spleen. In well-marked cases of pigmentation the spleen is discovered of dark brown or bluish black colour; which may be uniform or the parenchyma may be speckled. The organ is then soft, congested, and generally enlarged.

There are two morbid states incident to this viscus from morbus cordis, which exhibit different diseased conditions. In cardiac ischæmia the organ becomes dark, and it may be somewhat indurated, and the entirety of the parenchyma assumes that alteration of colour and consistency. In that infarction which is preceded by inflammation and ulceration of the endocardium, and therefore in connection with blood changes, defined patches are seen on the surface which are paler than other parts of the capsule, and which patches on section are wedge-shaped, the apex being in central direction, the base at the surface. The arteries supplying these circumscribed spots have been found blocked by ante-mortem clots. The vascular walls at length give way, blood is effused into the circumjacent substance, or the limited areas of such extravasations assume something of that apoplectic character which is known from a like cause to eventuate in the pulmonary parenchyma. Absorptional wasting, the formation of pus, or capsular peritonitis are apt to follow. It was the opinion of Rokitansky that the chief diseases of this viscus arise from anomalies in the blood and serum, or from certain dyscrasiæ, not well understood, yet which bear a remarkable and positive relation to the spleen. When speaking of secondary splenitis, caused by the absorption of poisonous inflammatory products, or affected in an analogous manner spontaneously, and showing the delicate reaction of this organ upon a morbid state of the blood, he says the formation of inflammatory spots is in every way remarkable. They are well defined, and occupy the peripheral portion of the organ, presenting a cuneiform shape, the base being at the surface and the apex pointing towards the centre.

That autho considers this form of its inflammation to be identical with pyæmic deposits, and to consist of the metamorphosis of an infected coagulum, within the channel of a vascular ganglion. These fibrinous blocks, to which reference has already been made, like apoplectic extravasations, have a tendency to deliquesce; but when they originate from cardiac emboli, as they sometimes do originate in this organ, there is not that proneness to rapid softening and disintegration which there is when pyæmia is the cause. The sudden arrest of nutrient blood to any particular area, as shown by Cruveilhier and Wharton Jones, at once produces capillary congestion, which is followed by the consequential and more cognizable phenomena of inflammation. Kirkes and Virchow pointed out that detached fibrinous vegetations in cardiac disease, and most especially in endocarditis, are swept on in the current of the blood until they block up an arterial branch, or it may be several branches, in some distant organ, as the brain, the lungs, the liver, the kidneys, or this viscus. Sometimes embolic infarctions have been found where no conditions could be detected to which the causation of emboli could be referred. The so-called arterial thrombosis might be the cause. When no valvular roughening nor any acute cardiac disease has preceded, ante-mortem coagula, from long enfeebled heart's action, might, as some pathologists have suggested, give rise to the phenomenon now more particularly considered. When these fibrinous pieces were deposited in the kidneys as the consequence of emboli or acute rheumatism, Rayer regarded the morbid condition as that of rheumatic nephritis. That which Rokitansky looked upon as capillary phlebitis is embolism, and the cuneiform blocks, when unassociated with greater evidence of pyæmia, may correctly be attributed to fibrinous obstruction. Dr. Wilks had under his care a young man in Guy's who died of ulcerative endocarditis. On examination after death, the spleen was discovered to be covered with lymph, which was evidently of recent formation. On section, the organ was seen to contain large abscesses of greenish glutinous pus, and which did not resemble softened fibrin, and the arteries running into the abscesses had plugs of the same granulation-like substance which was found in the mitral valve. The spleen is not the usual seat of pyæmic suppuration, but is more frequently the place where emboli give rise to the suppurative process when caused from cardiac disease. Sometimes it is difficult to say whether such depositions come from a pyæmic or an embolic cause. There is no doubt, also, that fibrinous clots may be produced in the arterial branches themselves, and that such pieces may plug up vessels of smaller calibre, and the same morbid results obtain. The consequences of thrombosis and embolism are on the whole very nearly the same, if not identical with those pathologic changes which obtain in secondary pyæmia. Still there are distinctions between these affections. In pyæmia there are generally purulent deposits in some other parts of the body, and not unfrequently the affection is obviously referable to a localized cause. There is, also, great tendency to suppurative decomposition. The proneness to destructive and degenerative change is more apparent than in mere embolia. When in secondary pyæmia the spleen is affected, the morbid conditions which inspection reveals are dark circumscribed extravasations of blood, which are of apoplectic character; they exhibit a great tendency to break down and degenerate into puriform collections, and there are generally some small abscesses to be found in the parenchyma. Murchison reports a case of embolism of the viscus produced by enteric fever, and in which dissection revealed two yellowish wedge-shaped deposits of the size of chestnuts, and he asserts that this kind of deposit is the most common in the spleen as the result of enteric fever. This physician also describes the same kind of pathologic appearance as following relapsing fever. A young man aged twenty died of this type of fever, and his spleen was found to weigh thirty-one ounces. The organ was soft except at either end, where there was a firm pale mass as large as a small orange, and exactly like the masses ascribed to embolism.

There is doubtless some similarity between the condition of the blood in scorbutus and in chronic splenitis. In both forms of disease the cicatrices of ancient wounds and old ulcers are apt to break open, and cutaneous injuries are difficult of healing. Graves mentions the tendency of chronic splenitis to give rise to suppurative ulceration of the legs, and he quotes from Aretæus, who in his time had not failed to observe the same peculiarity. Voight affirms the same to obtain in Bengal. The latter records that blisters and even leech-bites produce phagedænic ulcers which run on to a fatal termination. Twining gives similar testimony. He says patients are prone to sloughing ulcers from slight wounds and bruises; also that those peculiar characters of active inflammation,—and that constitutional energy on which the deposition of coagulable lymph depends, and by which we find injuries repaired, and the extension of ulceration as well as the progress of sloughing arrested on ordinary occasions,—seem to be in a great measure, if not entirely subverted. He remarks, too, that the blood drawn from patients coagulates imperfectly, and that the crassamentum was not unfrequently black and soft without exhibiting the buffy coat. On this particular Sir Ranald Martin thus writes:—"A wound or trifling abrasion, which at another time would escape notice, now becomes a foul and sloughing ulcer, owing to the depraved state of the blood, and the generally diseased state of the system hæmorrhages arise from slight causes, sometimes spontaneously; and so altered is the character of the blood, from the want of red globules apparently, that when performing surgical operations of immediate necessity at the Native Hospital of Calcutta, I always became aware of the presence of splenic disease on making my first incision, the hue of the blood being demonstrative of the fact." Muscular debility, impairment of the respiratory and assimilative functions, dropsies, and gangrene of the cheeks and gums, are the not uncommon associations of such depraved condition of the circulating fluids.

There is a large class of diseases of acute forms, which are not localized, in which the entire mass of the circulating fluid undergoes change, and in which, as above observed, the spleen in greater or less degree becomes implicated. In specific fevers such is eminently the case. Some morbid materials, it may be the matter of contagion, some miasma or virus, is imported into the system; it is infinitely multiplied in the blood, and certain constituents of the blood undergo transformation; and in such diseased condition of the circulating fluid new morbid products also become added to it. In the various kinds of continued fever, in the exanthems and periodic fevers, such changes very largely enter. Certain of the German pathologists have latterly attributed much importance to splenic enlargements when associated with acute febrile affections; and they regard such enlargement as an objective and a prominent fact which ought to count for much in the formation of diagnosis. Sir William Jenner, in this country, has also insisted on the same fact. Friedreich of Heidelberg is of opinion that the tumefaction of this organ in numerous acute febrile diseases cannot satisfactorily be accounted for on the assumption of simple fluxion, and he says we must look for other causes for the great hyperplastic proliferation which is, under such circumstances, not unfrequently present in the pulpous elements of the viscus. In the class of cases now particularly instanced, there is, doubtless by the importation of foreign matters into the blood, special irritation instituted in the splenic pulp. This writer insists that such changes of volume in the spleen would often justify us in deciding as to the infectious nature of a malady when the etiology might be otherwise obscure. He goes on to say, " The readiness with which the spleen reacts to certain substances of an injurious nature present in the blood is explained, not only by the copiousness of its blood supply, but also by its anatomical peculiarities. We know that the arteries in the interior of the spleen break up into very numerous, extremely fine branches and capillaries, perforated by stomata, that the blood passes from the vessels into wide spaces destitute of walls (the intermediary blood canals), from which the veins begin to arise in like manner, principally as cribriform canals. The blood flows with extreme slowness through these wide intermediary canals, and both here and in the cribriform perforations of the capillaries is in intimate connection with the lymphoid cells so susceptible to irritation, and the delicate, fibrous network of the pulp. Hence it follows that matters of an injurious nature infecting the blood are extremely liable to be retained in the spleen, to accumulate therein, and to act with peculiar intensity as excitants to the splenic tissues." The authorities on this subject point out with much assurance the regular occurrence of splenic tumour in enteric fever. In doubtful cases of enteric they would regard this physical sign as decisive of the diagnosis. Türgensen, in what he terms *typhus levissimus*, looks upon the presence of splenic enlargement in doubtful cases as sufficient, in conjunction with other symptoms, to indicate the true nature of the primary malady.

It is not, however, supposed that there is an exact correlation between the augmented volume of the organ thus increased and the gravity of co-existent febrile phenomena. The spleen, they say, may be large in the mildest forms of enteric, and only moderately augmented in the severest examples. The resisting power of the constitution against the irritative properties of the contracted infectious matter, they affirm, will account for this disagreement in the greater or less obstructive effects, and the more pronounced or moderate hyperplastic turgescence. The spleen, it is said by the authorities to whom reference is now made, thus becomes acted upon at the very outset of the enteric attacks, because the specific poison of that fever has a remarkable tendency to morbidly influence this viscus. Some of these writers aver that splenic tumour supervenes even during the incubative stage of enteric, and that it is the last amongst those organs usually affected in that complaint which returns to the normal size and condition. And relapses in enteric are said to be more probable when, during convalescence, the spleen is slow in its diminution. Sometimes the swelling will be to such an extent that it will project to a considerable distance below the costal edge. It is to be held in mind that on making an examination of the state of the spleen in enteric fever much fallacy may result from the condition of tympanitis, and there may be decided enlargement, whilst the region when percussed is resonant. Careful palpation is then our safest guide. Türgensen says enlargement of the spleen occurs probably in all cases, and is a constant symptom even of the mildest enteric. Out of eighty-eight cases, in seven only was enlargement not recorded; hence it was recognised, therefore, in ninety-two per cent., and, he remarks, it was often demonstrable very early, not rarely on the second or third day of the disease. Sometimes a long period elapses before the hyperplastic elements undergo solution and absorption, and before the contractile functions of the organ became fully restored; and such tardiness in a return to the normal configuration is doubtless not only dependent upon the presence of new materials thrown out, but also consequent upon the grave impress being made by the specific poison upon the splenic plexus. In diphtheria, as in enteric fever, the spleen is said not uncommonly to be increased in its diameter. Birch-Hirschfeld says splenic tumour occurs as the rule in hæmorrhagic variola, and Trojanowsky attests to the same coming on in scarlatina and measles. In the first-named he had several times been able to demonstrate clearly the existence of a splenic tumour in the irritative stage, and in the majority of instances after the appearance of the eruption. During an epidemic of measles which raged in Heidelberg in the winter of 1873-4, Friedreich repeatedly convinced himself of this complication in that exanthem. In erysipelas, in acute coryza accompanied with febrile phenomena, and in that masked, wandering, and which has been termed serpiginous form of pneumonia as contra-distinguished from the better defined and more recognised croupous inflammation of the lungs, these physicians affirm that splenic tumefaction is usually to be discovered, and that the proneness of this viscus to exceed its natural size in these respective ailments affords proof of their primarily infectious character. If the foregoing statements, which these writers have with such prominence put forth, are found to hold good as the rule and not exceptionally, splenic tumour would often and very properly be sought for as an aid to diagnosis in many otherwise ambiguous instances of infectious diseases. During the first six or eight days after the invasion of enteric fever, when the true nature of the complaint sometimes puts on such conflicting appearances, and when there often is to the most experienced eye so much difficulty in its recognition, an additional fact like that now spoken of, to aid in our decision and direct our judgment aright, would be of the greatest value. "I feel convinced," says Friedreich, "that a careful consideration of the condition of the spleen would enable us to recognise the infectious nature of many disorders which present themselves as apparently merely local affections, and that the group of the acute infectious diseases would be thereby considerably increased." That the spleen is frequently involved in the various forms of febrile affection, and to a greater extent than was formerly supposed, can hardly be disputed, but that it is so very commonly the seat of lesion, as these pathologists insist upon, requires still a greater accumulation of proof.

If the endeavour were made to enter into an explanation of these etiological conditions whereby the spleen is so prone to become the seat of lesion in various infectious diseases, such would be to discuss the fundamental agencies of contagion and miasma, and in much that could be advanced to pass into the dubious domain of hypothesis. Late investigation has, however, elicited not a little which seems in no slight degree to harmonise with theories which have long been put forth, and which have been believed to be explicative of clinical phenomena. If we are to endorse the notion of attributing to the origin of many acute infectious ailments the absorption of a *substantia viva* into the body, the primary blood contamination can then be understood. The more recent researches of pathology tend to give credence to the supposition of minute low vegetable organisms constituting the cause of that class of diseases to which reference is now particularly made, and this view in some degree gives confirmation to the doctrines which found favour with the older physicians, and who held the idea of a parasitic origin of diseases. The generic group of these organisms which has been named schizomycetes is said to possess species called bacteria, bacteridia, vibriones, spirillæ, and other distinguishable kinds of minute entities, and it has been imagined that each of these microscopical organisms give rise to a distinctive train of morbid actions. Davaine showed malignant pustule to be preceded by bacteridia in the blood; Klebs believed from certain experiments that the microsporon septicum could cause continued fever; and Letzerich has maintained that the schizomycetes discovered in the blood in diphtheria are peculiar, and differ from the other and recognised forms of those in the lowest types of animal life. If, then,

te infectious diseases are to be referred back to the presence of
rying and dissimilar microscopic organisms, much light may be
d to be thrown on the origin of what is termed the great class of
otic diseases, and which destroy a sixth part of the population.
t is the opinion of those investigators who have closely studied this
ject, that morbid phenomena are not to be referred merely to the
sence of these corporeal particles in the blood and their mere
ysical effects, but rather to the irritating virus which they secrete,
d the power which such virus possesses of instituting diseased
ion; again, they may, it is possible, abstract from the blood certain
stituents essential to nutrition and the normal forces of vitalism,
d hence another cause favouring morbid processes. The function
d nutrition of organs and tissues having become implicated, a
ater or less amount of disorder would be given to the general
stem. And as it has been said that some particular parts are more
ble to become the habitation of schizomycetes than other parts, a
cal prominence would thus be conferred to the disease. The vascular
paratus of the spleen has been said, as above remarked, to favour
e concentration for these minute and organised germs. Coze and
ltz in small-pox detected the largest number of bacteria in the
pleen; Birch-Hirschfeld saw micrococci in large accumulation in
hat organ; and Grimm discovered in animals which died from malignant pustule, bacteria very numerously in this viscus. If the views
above cited are to be accepted, and if these organic particles largely
accumulate in the spleen, then their poison would also be concentrated
in the protoplasm of the cells of the pulp, and thus an explanation is
afforded of the early manifestation of splenic tumour, and, too, a
reason is given why this organ is slow in its return to a normal
state.

In pyæmia, icorrhæmia, and septicæmia, and in purpura and scurvy, lesions of the blood constitute the cardinal symptoms. In nearly all, it may perhaps be said in absolutely all, of these named, there is decrease of fibrin and excess of red-corpuscles, and, as a consequence, one or other form of hæmorrhage often comes on. When in these complaints this organ is in secondary manner affected, and its congestion gives rise to hæmorrhage, the loss of blood may be from epistaxis, or hæmatemesis, or by the passage of blood by the bowels. The tense and fibrous tunic of the viscus prevents its being relieved by serous exudation, as the lungs are relieved by effusion into the pleura, the peritoneum by ascites, or as a congested liver and a congested gastro-intestinal surface are relieved by serous diarrhœa. Sometimes the hæmorrhage by epistaxis, or by hæmatemesis, may be to an alarming degree or it may be fatal. A young man of pallid and anæmic aspect was some time ago admitted into the Tunbridge Wells Infirmary under my care. He stated that he had gradually lost flesh and strength, and that he had been obliged to relinquish his ordinary employment, which was that of a farm-servant. He was twenty-two years of age, the physical signs of the thorax were normal, there was no hepatic enlargement nor had he ever been jaundiced. On careful examination the splenic dulness far exceeded the normal area, the edge of the organ could be felt depending below the left cartilage, and it extended horizontally nearly to the mesial line. I carefully mapped out the lines of dulness on his admission. He was treated with iron, sulphuric acid, and bitter infusions, the bowels being moderately acted upon by aloetic purgatives. He was discharged at the end of a month, looking and feeling better, and on percussion the splenic dulness occupied a decidedly diminished space. After having left the institution ten days profuse loss of blood came on from the mouth and nose. The hæmorrhage continued at intervals during the three succeeding days. He lost two quarts of blood, and sank from exhaustion. Sir T. Watson says he has more than once seen hæmatemesis in connection with splenic enlargement, and he quotes from Morgagni, who relates a case wherein, after repeated attacks of hæmatemesis the patient sank, and on cadaveric inspection the viscus was found to weigh five pounds and to be gorged with dark blood. He also cites from Frank, who gives the history of a patient who had vomiting of blood, and the organ weighed sixteen pounds. That case had, however, evidently been one of leucocythæmic enlargement, as we now know that in white-globule-blood hæmorrhage is liable to occur. Latour gives several cases which he had selected from various authors, in which splenic enlargement was followed by hæmatemesis. An illustrative example of that which is now asserted lately occurred to me. A lady who has a very large spleen which almost fully occupies the left hypochondrium, and the anterior edge of which extends to the median line, and whom I now professionally see from time to time, had several months ago a terrible attack of hæmatemesis. She then lost within twenty-four hours between five and six pints of blood, and the exsanguination became so extreme that she had syncope. From my first acquaintance with this patient she looked pale and anæmic, and there were all the external appearances of splenic disease in addition to the more conclusive testimony of markedly existent physical signs. The hæmorrhage has not returned. This large loss of blood seemed for a time to render the tumid spleen of less volume.

This gland, as before remarked, is very importantly concerned in the formation and disintegration of blood-globules, and if the chemical constituents of the blood are changed, it cannot normally perform its office, and as a consequence must necessarily of itself become diseased. Its enlargement and loss of vital adhesion are its most common alterations.

ACUTE SPLENITIS.

There may be acute inflammation of the spleen, as there may be acute inflammation of any of the other solid viscera, but the true sthenic type very seldom occurs, at least in this climate. In malarial countries it is not so uncommon, and more especially in tropical regions. When it is observed in this country it is generally referable in the extension of the inflammatory process by continuity of structure and proximity of position, as when the serous coverings of adjacent organs and parts are inflamed; or it may be produced by external violence. From such causes it may obtain at any age. M. Huguier gives an example of acute splenitis in a child not four years old.

CAUSES.—In addition to the causes already named, sudden distension, and stretching of its fibrous capsule under the malarial influence; exposure to a lowered temperature after hot and sunny days; long and fatiguing marches; great bodily exertion, such as protracted journeys in damp and marshy districts; hæmorrhagic infarctions as connected with hyperæmia; emboli originating in left heart valvular disease; and the suppression of accustomed discharges as hæmorrhoids and the catamenia have been regarded as the etiological conditions. Long says inflammation of the uterus has been found to be succeeded by acute splenitis. Hæmorrhagic infarctions occurring during the progress of infectious diseases sometimes constitute a cause. Intemperance may also be named.

SYMPTOMS.—According to Grottanelli, Bree, Craigie, and others, acute splenitis is ushered in by shivering and chilliness alternating with flushes. There is a sense of tension, weight and dragging in the left hypochondrium; pains are felt deep in the side, sometimes of sharp and stabbing character, which radiate into the epigastrium, or round to the spine, or extend to the left shoulder, breast, and clavicle. Sickness and vomiting may supervene, the ejected matters being a biliary mucous fluid and occasionally mixed with blood. Sometimes the blood vomited may be large, and when there is sanguineous extravasation into the stomach the stools are necessarily darkened. There may be some hæmoptysis or epistaxis. Twining says that inflammation of the serous coat sometimes takes place without much enlargement of the viscus and that the symptoms greatly resemble those of pleuritis. If the peritoneal covering of the diaphragm be involved, cough and dyspnœa are prominent symptoms. Thirst, loss of appetite, furred tongue, confined bowels, scanty urine, heat of skin, and flushed countenance obtain, and in the evening the general conditions of pyrexia assume more or less of exacerbation. Sometimes hiccough, tympanitis, diarrhœa, delirium and exhaustion come on, and the patient will die in the course of a week or ten days. The pulse is quicker than natural, but its acceleration is not always in a ratio commensurate with the severity of the concomitant symptoms. The pulse may not exceed 90, and the temperature be only 100° F. even when the case is progressing to a fatal termination. Sudden and diffuse peritonitis may come on when all the cardinal symptoms rapidly become more grave. Certain of the older writers have endeavoured to account for the comparatively slow pulse by attributing such peculiarity to the distensile and accommodating qualities of the organ, by which they conceived it to be less prone to vascular obstruction and blood stasis, the notable conditions of inflamed parts, and which so potently react upon the circulatory and nervous systems. The patient cannot lie on the right side, and whenever he attempts to do so the dragging pain is much increased. When purulent formations take place there are rigors and flushings, and a throbbing in the splenic region, and the patient lies partly on the back and partly on the side. In the worst cases of acute splenitis there may be black stools, these being voided unconsciously. In those examples which pass on to a favourable termination there is the gradual mitigation of pain; the patient is by his feelings less restricted to one position in bed; the breathing is easier; the pulse and temperature begin to fall; there is not uncommonly a copious perspiration; deposits are seen in the urine; diarrhœa may come on; the tongue is more moist and less furred; sometimes there is some loss of blood by hæmorrhoids; and in females the catamenia may appear; the evening exacerbations are not so pronounced, and the sleep is more continuous. In the course of a few days after such decline of the more prominent symptoms the physical signs likewise indicate improvement.

Diagnosis.—When the inflammation of the peritoneal covering has been established, the fibrous capsule and the substance of the organ also become affected; there is then some enlargement, and palpation will generally discover the lower edge of the organ ranging with or depending below the costal cartilages. The complaint may be distinguished from hepatitis from what has been above said relative to the physical signs, and accurately observing the seat of pain; by the absence of jaundice and the colour of the excretions; from inflammation of the stomach, by the sickness not being a cardinal symptom; by pressure being borne at the epigastrium, and by remembering that idiopathic gastritis is very rarely seen in this climate. It may be known from pneumonia and pleurisy in the lower half of the left thorax by the non-existence of those stethoscopic signs so characteristic of the respective affections; it differs from nephritis in the locality of the pain, by its not following the course of the ureter, by there being no retraction of the testicle, and by the analytical and microscopical appearance of the urine; and from ordinary peritonitis by the clinical history of certain negative facts, by the state of the pulse, and the tenderness not being diffused over the abdomen. Its resolution is judged of by freer flow of urine, moist tongue, improved circulation, less pain, cooler surface, and the more natural appearance of the features.

Treatment.—In those cases which may be regarded as being of the sthenically acute type, when the vital powers are unimpaired, as evinced by strong and quick pulse, heat of skin, thirst, furred tongue, accelerated breathing, cough, and pain on hypochondriac pressure, local blood-letting may be employed. If the tenderness be not too great cupping may be had recourse to, but if the local pain be considerable leeches should be ordered. The part having been then well fomented, a large linseed-meal or bran poultice should be applied; this should be covered over with cotton-wool, and then a piece of oiled silk should be placed over, extending fully beyond the margins. The fomentations may be with poppy-head decoctions. In some cases terebinthinate epithems are of great service. When the acuter symptoms and the symptomatic fever are subdued, stimulating liniments, blisters and moxas are of use. Aloes, antimonials, and neutral salts are at the first beneficial. Afterwards the compound extract of colocynth, the compound jalap powder, and various preparations of senna, scammony, or rhubarb are appropriate remedies. Mercurials should, except occasionally in union with purgatives, be avoided. When there is sympathetic vomiting the carbonates and bicarbonates of the alkalies may be administered. A carefully regulated diet and rest in the recumbent posture should also for some time be observed.

CHRONIC SPLENITIS.

There are many indications in common to both the acute and chronic forms of the disease. In chronic splenitis the degree or intensity of the affection is most varied. It is generally the complication of ague, and is most frequently observed in the malarious districts of the East. It is often a consecutive complaint, and is seen mostly in conjunction with intermittents and remittents, or as associated with hepatitis and dysentery. In the continued fever of this country it is occasionally noticed concurrent with or as the sequel of that disease. The more obvious conditions of its covert or low form of inflammation are sometimes preceded by congestion. And the acute inflammatory state may be succeeded by the chronic. In some instances the envelopes become inflammatorily affected, when degenerative products may in the course of time be found in the tunics, as the cartilaginous or ossific conversions; or greater or less amounts of purulent secretion may be formed in its parenchyma.

CAUSES.—The causes of chronic splenitis are those etiological conditions which have already been spoken of when treating of the etiology of splenic diseases generally. By far the most common cause which can be mentioned is the endemic influence, and most especially the malarial exhalations which are so usually given off in fenny districts and in tropical countries. In the East and West Indies, and in other of our colonial possessions, where intermitents and remittents are so commonly met with, this affection is very prone to occur, not only in the white but in the dark races, and particularly among young people, and in the children of European residents. The fluxionary hyperæmia to which this gland is subject in periodic fevers, and the repetition of surcharges of blood, induce a proneness to a low form of inflammation in its structures, and the inflammatory phenomena thus set up are often insidious and protracted. The more cognizable conditions of the sthenic or acute affection may as remarked become chronic. In the various forms of continued fever this organ may in consecutive manner become chronically inflamed, and in the relapsing type this observation more especially holds good. In the organic changes of other organs this gland sometimes becomes chronically inflamed. In cirrhosis of the liver, and where from any other cause there is obstruction of the portal vein, such may be the case. Wherever circumstances may obtain which mechanically obstruct the hepatic circulation a low state of splenitis may occur.

SYMPTOMS.—The more prominent symptoms are a feeling of weight and heaviness in the left hypochondrium, which is aggravated by lying on the right side. There is uneasiness on pressure over the region. The patient describes the pain as dull, obtuse, and aching, and it extends, as Morgagni observed, towards the spine. There is a distressing sense of fulness and tension at the epigastrium, which is more prominent and rounded than normal. The sleep is broken and fitful, and disturbed by unpleasant dreams; there is more or less of dyspepsia, and there is often a light-coloured glassy tongue; and when the diaphragmatic covering is affected the breathing is hurried and shallow, as in the acute types; added to which there is a dry, teasing cough, and there are also anorexia, flatulency, torpid bowels, scanty urine, dry skin, and furfuraceous desquamation of the cuticle; and acceleration of the pulse comes on more towards the latter than the earlier stages. This, however, is not an invariable symptom. Bree says the pulse is slow during the earlier or congestive stages, and only becomes quick when the tunics are at length painfully distended; he also adds that the gland may for months be turgid without giving rise to symptomatic fever, and that a moderate degree of fever is indicative of an effort on the part of nature to resolve the disease. In the evening, or during the night, there is exacerbation. In this condition of the organ, blows, falls, and external injuries are apt to be followed by acute splenitis. When the complaint is of long continuance, when it passes into what may be termed the third stage, when remedies have proved inoperative, and when change of climate cannot be obtained, still graver conditions become apparent. There is then increased debility, the legs are smaller, the loss of flesh is more marked, convulsive asthmatic attacks supervene, and the enfeebled heart is exemplified by fits of palpitation which follow exertion. The organ extends below the false ribs, and often obliquely towards the umbilicus. The anæmia, blanched sclerotic, and tawny countenance proclaim augmented deterioration of the circulating fluids and depressed secretion, whilst the progressive emaciation, ascites, hectic, dyspnœa, singultus, vomiting, and diarrhœa usher in the fatal termination. Blood mixed with matter ejected from the stomach, or with the alvine evacuations, not infrequently occurs, giving temporary relief, and the sanguineous discharge would seem for a time to ward off the mortal event. Such are the main symptoms of chronic splenitis, and more especially as the affection prevails in malarious countries.

Diagnosis.—When the patient complains of a dull, aching, dragging weight at the base of or below the left posterior thorax, with pains extending through into the back or radiating up into the left shoulder; when he lies on the left side or with some tendency to reclination on the back; when there is the clinical history of a residence in some aguish district, when the features look dusky, yellowish and anæmic, when the lips and gums are bloodless, or when these symptoms have come on gradually, chronic splenitis may be suspected. Physical examination will pretty certainly confirm the suspicion. When the disease obtains, palpation and percussion discover enlargement of the viscus. The normal lines of dulness are in varying degrees exceeded, and the free border of the gland may be felt at or even below the costal edge; nor should it be forgotten, what has in this article already been insisted upon, that the viscus in health cannot be touched with the fingers. The patient in chronic splenitis bears examination much better than he can bear manipulation in the acute form. He generally can alter his position in bed, move the trunk, and make full inspiration and expiration without any great increase of pain. He does not complain of those stabbing and lancinating pains which are so symptomatic when the serous and fibrous envelopes are inflamed. Dulness, heaviness in the side, and aching, are the terms the patient uses as descriptive of his ailments. When dyspnœa and pleurodynia are present, it is presumptive that the upper part of the organ is more particularly enlarged and the seat of disease, and that pressure on the diaphragm has affected that organ. Chronic splenitis, with enlargement, which occurs as the rule, may be known from pleuritic effusion by negative as well as positive facts. There is not the intercostal stretching and bulging, nor that smooth rotund configuration of the chest-wall, which denote the accumulation of fluid. Percussion in effusion elicits an absolutely dull, dead sound, which to the practised ear is markedly different from the kind of dulness produced by any other physical and pathologic changes. Higher up there may be friction sounds or ægophony. Again, in pleuritic affections there is the characteristic cough in the earlier stage of the complaint, and when there is the accumulation of liquid the dyspnœa is a notable condition. In pneumonia the dulness is not so absolute, it extends over a larger area, the breathing is quick and short, the countenance often has a venoid hue; the aspect, pulse and temperature point to the pneumonic nature of the ailment. In inflammation of the peritoneum the decubitus, pulse, abdominal distension, the more extended tenderness, the drawing up of the legs, and thoracic breathing are significant of peritonitis. The peritoneum may become locally inflamed in splenitis, but in such cases the tenderness on pressure is circumscribed, there is not much abdominal distension, and the patient can turn towards the side, and he does not lie with his legs drawn up. The pain on pressure is confined to the left side. In nephritis the ordinary urinary tests and microscopical examination are our guide. In encysted kidney accurate manipulation will enable us to discover the one affection from the other. Ovarian tumour can hardly be mistaken for the complaint. Ovarian enlargement is first felt lower down in the abdomen, and there is generally resonance between the costal edge and the ovarian dulness. When there is a purulent collection in the splenic substance, the formation of matter is decided upon by the tenderness on examination; there are rigors, hectic towards evening, and in the course of time fluctuation may possibly be detected. Impacted colon may be judged of by its irregular contour, the sulci which can generally be felt, and a large enema either removes the tumour or alters its configuration. Malignant growths of the omentum are harder, flatter, broader, and their edges are less defined, and the clinical history will conduct to the correct conclusion.

TREATMENT.—When there is great and manifest enlargement, when the pain and aching are considerable, when it is believed there is some peritoneal tenderness, and when the affection occurs in a person of strong and unbroken constitution, leeches may be applied, and these should be followed by a large linseed poultice. When local blood-letting is not had recourse to, and as it seldom will be required, fomentations with strong poppy-head decoctions are soothing and beneficial. A large bran or linseed-meal poultice applied in a flannel or muslin bag is excellent practice. The poultice should be covered with cotton-wool, and over the latter should be spread a piece of oiled-silk sufficiently large to extend beyond the margins. These poultices should be frequently repeated, and never allowed to remain on when they are becoming cool. When the symptomatic fever is subdued, stimulating liniments, such as the soap, turpentine, and camphor liniments of the Pharmacopœia, or these may be combined, and the opium and belladonna liniments may be added. Terebinthinate epithems, and sinapisms are often of much service. Some authors recommend drastic purging, to be for some time continued. In the chronic inflammatory state there is no doubt that purging is one of the most successful and best remedies. Hippocrates and Celsus had observed the curative tendency of dysenteric complaints in splenic disease, and this salutary effect of increased action of the bowels can be well understood when considered anatomically and physiologically. Engorgement of the splenic veins produces vascularity in the vessels of the descending colon and rectum, because it engorges the inferior mesenteric, from which those parts of the alimentary canal derive their blood, and the hyperæmic mucous membrane constitutes the proximate cause of the dysenteric symptoms. Aloes, antimonials and neutral salts, by determining the blood to the pelvic and abdominal vessels, produce the best results, and whenever the organ is relieved by nature or by art it is mostly by the vessels of the lower viscera receiving more than their normal quantity of blood. Certain Indian writers are of opinion that in enlargement of the Spleen a prompt antiphlogistic treatment should at once be employed. And they give it as the opinion founded on extensive practice that local blood-letting, epispastics, or counter-irritants, ought to be applied, and that brisk purgatives should be combined with such treatment. If antimony and salines be too long used, debility ensues. Recourse may then be had to the compound pill or the extract of colocynth, the compound jalap powder, and the various preparations of scammony, senna, and rhubarb. To act on the radicles of the portal vein is to relieve splenic as it relieves hepatic congestion. The combination of calomel may be advisable when there is a turgid condition of the liver. The employment of mercury, otherwise than in union with purgatives, cannot be too strongly reprobated, as all authorities are averse from its use except in the manner described. Lamentable effects have resulted from the way in which mercurials were formerly administered in splenic complaints; and from what has been said in this article relative to that dyscrasia of the fluids so commonly the accompaniment of the diseases of this organ, it is obvious that mercury is not only an unsuitable but a very deleterious agent. Some Indian writers have drawn terrible pictures of its ravages, and are most emphatic in its denunciation. A cloth dipped in dilute nitro-hydrochloric acid, placed over the splenic region, and then covered with an emollient cataplasm, is a means for adoption. The nitro-hydrochloric bath in the advanced and improving stage of the disease will expedite recovery.

The sulphate and tincture of the perchloride of iron in union with purgatives, more particularly the aloetic preparations, have been, and deservedly, lauded. When there is chronic splenitis with enlargement, amenorrhœa and chlorosis, iron in some form should be given. Twining speaks of what is termed the splenic mixture as prescribed in India, which consists of jalap, rhubarb, calumba, ginger, cream of tartar, and sulphate of iron. The nitric and hydrochloric acids with infusion of chiretta constitute a good medicine. A sea-voyage and change of air will often expedite the cure. M. de Parville has recently propounded the idea that chalk may be regarded as prophylactic, if not as a more absolute remedial agent, in splenic complaints. It was observed that during the prevalence of a splenic disease which affected sheep and cattle, that those animals which licked white-washed walls had in a great measure an immunity from the complaint. This effect of the carbonate of lime requires, however, in the human subject more evidence than has hitherto been adduced.

DISEASES OF SPLEEN IN CHILDREN.

Lesions of this organ in children are by far most commonly seen in those who have lived in low and damp situations, or who have been brought up in malarious districts and tropical climates; we sometimes, however, meet with splenic diseases in children in this country, and more especially in dispensary and hospital practice, where poor and insufficient food, bad ventilation and humid houses have exerted a predisposing effect. Infants of a few months old sometimes have enlargement of this viscus, but in such instances the enlargement can hardly be referred to those more general and extrinsic conditions which so prominently enter into the etiology of splenic affections in the adult.

CAUSES.—The causes of the morbid changes in this gland in children and the young are as the rule malarial emanations. In aguish parts of the country, the kind of complaints now more particularly considered are not unfrequently seen. Protracted lactation may predispose to splenic ailments, but they are by far most usually met with as the accompaniments and sequels to intermittent and remittent. types of fever. In certain affections of the liver which prevail amongst children the Spleen is apt to take on a congestive form of disease. The portal circulation being obstructed, fluxionary hyperæmia of this viscus may occur, and hyperplasia of the cellular elements at length take place. Quite independent of the malarial influence, the disease now known as leucocythæmia may come on in infancy and childhood, the causes of which are to be regarded as constitutional and not owing to unfavourable hygienic circumstances. In some instances, in children, the cause of its pathologic changes is degeneration of the walls of the vessels and the cell elements of the pulp, which go to form the lardaceous change, like to the lardaceous change in the liver. It may be in association with tuberculosis. In rachitis and scrofulosis a similar diseased mutation occurs. In hereditary syphilis this organ may present that amyloid degeneration which is now known to be so characteristic of this specific disease.

SYMPTOMS.—When the affection is marked, the little patient lies on or towards the left side. Percussion discovers an increased area of dulness, and palpation detects the depending organ on or below the costal edge. The initiatory symptoms are restlessness, peevishness, anorexia, sleeplessness, and a disregard for wonted amusements. . There is a loss of flesh and strength, the child becomes pale and sallow, the lips and tongue look bloodless, and the conjunctivæ are blanched. The skin is harsh, hot and dry, the pulse often quick and feeble, and there are giddiness, headache, and palpitation. Pain is readily produced on pressure below the false ribs, the knees are often drawn up, and the trunk, as the little patient lies on his left side or towards the bed, is curved. The bowels are generally costive, and the urine is pale, and there is an obvious increase of the pyrexial symptoms towards evening. Hectic, dyspnœa, ascites, diarrhœa, and sometimes œdema of the legs, and a greater or less degree of anasarca, supervene towards the end. In rickets, scrofulosis, and in leucocythæmia the organ is large and impending, and it becomes the largest in the last-named complaint. In these affections the patient's aspect is anæmic, yellow and waxen, but the symptomatic fever is much less, or may not obtain, nor are there the evening febrile exacerbations. Not unfrequently the liver is also large, so that the lines of dulness on both sides are much extended. When the hepatic affection is marked, the superficial veins of the abdomen are apt to become full and turgid. With these forms of splenic disease there are petechial stains on the surface, and grave or even fatal epistaxis or hæmatemesis may come on. In syphilitic cases, from hereditary taint, when there is splenic enlargement, other and co-existent symptoms are observable. Coryza, cutaneous eruptions, and ulceration about the angles of the mouth are not uncommon. A yellowish ichorous discharge comes from the infant's nostrils, the lips are fissured, the Meibomian glands secrete pus, and often red, coppery spots can be seen on the skin. In such instances gummatous deposits are apt to be formed in other of the solid viscera, as well as in the Spleen.

Diagnosis.—The rules to be observed in diagnosticating splenic affections in children are much the same as those to be noticed in judging of the maladies incident to this organ in the adult. The clinical history aids in our decision, and the fact of residence where ague has prevailed at once points to the probable nature of the complaint. The physical signs in the child are easier of interpretation, more distinct and definite than they are in those who are older. The abdominal and thoracic walls are thinner and more resilient, and the internal parts can with less difficulty be felt. By careful examination of the chest, diseases proper to that cavity can with much certainty be excluded. Palpation, as above remarked, finds the edge of the gland and accurate percussion defines the extent of its increase. Jenner says, "If the fingers of the right hand are placed under the child's twelfth rib on the left side, and the fingers of the left hand a little to the left of the middle line, between the navel and the ensiform cartilage, and a pressure be made backwards and forwards, the Spleen may be readily felt." The front edge is more defined than the front part of a large kidney, and the dulness in a large kidney extends fully up to and under the spinal muscles. The tumour of psoas abscess does not reach so high up either as the Spleen or a large kidney; it is circular, and the tenderness on digital examination is very great, and as the disease progresses the secretion gravitates downwards. The enlargement of this viscus is very generally the accompaniment or sequel of intermittent or remittent fever, and the symptoms of those diseases will without difficulty be recognised. In abscess there is more localised tenderness over the splenic region, there are rigors, and towards evening the little patient becomes flushed, hotter, and more restless. In leucocythæmia the organ may become so large as to apparently occupy half the abdominal cavity; there is then a markedly anæmic condition, and the abdominal veins are sometimes distended and tortuous. Syphilitic enlargement will be decided upon from what has already been stated when describing the various symptoms of the diseases of this viscus.

TREATMENT. — The treatment of splenic diseases in children should be on those more general and recognised principles which are to be observed in the adult, and which have already been given in this article; and also, it need hardly be remarked that much exactness is always needed in prescribing for infants and young children, nor is it always safe to depend upon the doses which are arbitrarily fixed in posological tables; for it must be borne in mind that susceptibility in the young, with regard to remedies, is not unfrequently very pronounced. The duration of the ailment, and the general strength of the little patient will greatly determine the kind and amount of agent to be employed. If the child should evidently labour under acute pain; if the pulse be quick, the respiration accelerated, the temperature high, the skin hot and dry; if additional distress be caused on the movement of the trunk, or the inclination of the body towards the right side, and if a long breath cannot be drawn, and the urine be scanty, the bowels confined, the tongue coated, and there is much thirst, there will doubtless be more or less of acute inflammation. Not only may the fibrous and serous covering of the gland be inflamed, but the inflammatory action may have extended to the diaphragm, or there may be more or less of peritonitis. Fomentations and poultices should then be very sedulously applied, and if the acuter symptoms do not yield to their employment, leeches may carefully be used. Purgatives of senna, rhubarb, or some of their respective preparations, or some of the neutral salts, should be given. The hyperæmic distension by undue stretching excites the inflammatory phenomena, and to lessen the surcharge of blood in the gland is our aim; hence by acting on the bowels and thus unloading the venous radicles of the portal vein the splenic turgor becomes in an indirect manner lessened. A febrifuge may be given composed of the solution of carbonate of potash saturated with citric acid and flavoured with the syrup of mulberries; or the nitrate of potash dissolved in water, made palatable with the syrup of oranges or the syrup of lemons, form another mixture which children will readily take. The acetate liquor of ammonia with some agreeable addition is a good medicine. When the symptomatic fever has declined quinine is then imperiously demanded. In the chronic, sub-acute and more congestive forms of the affection, purgatives with senna, rhubarb or scammony, some preparation of aloes, in conjunction with iron and sulphuric acid, should be prescribed. The nitric acid bath, or nitric acid applied locally (always taking care that this drug should, when thus rendered available, be sufficiently diluted) are not unfrequently of much service. When the complaint has evidently resulted from periodic fever, removal to another district or another climate is a desideratum. In the leucocythæmic form iron is the remedy upon which the most reliance can be placed. In splenic enlargement of infants caused by hereditary syphilis, small and continuous doses of the ordinary pharmacopœial preparation of mercury and chalk, and afterwards the iodide of potassium, constitute the best and most effective treatment. In all the varied forms of splenic diseases in children much attention should be paid to the diet. Articles of food should be selected which are light and nourishing, such as are blood-making and digestible. In winter and cold weather flannel underclothing is of great importance.

ABSCESS OF SPLEEN.

Abscess of this organ is an affection which not unfrequently ends fatally. It is sometimes very insidious, taking place without any other symptoms than those of bad health and wasting. Upon enquiry it is generally ascertained that the patient has been subjected to the endemic influence of periodic fever, and that there has been pyrexia. It may be the result of acute splenitis, but it is more frequently caused by the sub-acute and chronic forms of inflammation. When the pus is in the centre of the gland, its diagnosis is most difficult, and it is only when it painfully presses upon the membranes that it can be detected. Shiverings, hectic, evening exacerbations, the general irritation caused by purulent absorption, and restricted secretions suggest its presence. Abercrombie gives an apt illustration of its covert progress in the case of a gentleman who for six months pined and wasted away without the exhibition of any local symptoms, save an insignificant catarrhal affection, and it was found on inspection that this organ contained a large abscess. Craigie bears like testimony, and observes that the most perplexing part of the semeiography and symptomatology is, that these collections give no evidence of their presence until by their size they painfully stretch the viscus and press upon surrounding organs. Heusinger and Schmidt say the same. Twining and Voight assert that splenitis rarely runs on to suppuration in India. When pus is formed in the organ, it is always as a rule the consequence of inflammation. Pus may be collected, however, in this viscus as it may be collected in the liver, in consequence of injuries in remote parts and when pyæmia is present in the system. Nor does the suppurative process exert that deleterious and destructive effect upon the structure of the organ which might be supposed. The filamentous tissue may be bathed in pus for a long time without injury. The suppurative formation in one or more sacs may be borne for a considerable period, and the parietes of such sacs at length become fibrous, cartilaginous, or even osseous, and the contents may, by partial absorption, be rendered a greasy, calcareous pulp. Much more frequently the inflammation by peripheral pressure of the sac extends towards the surface when the investments give plainer indications of lesion. There may then be adhesion to the diaphragm or abdominal wall, or to the stomach, colon, or ileum. Fluctuation may be felt and external pointing observed, just as these conditions are apt to be presented when there is abscess in the liver. The matter may be discharged in various ways. It may be poured into the abdominal cavity, when flagrant and fatal peritonitis rapidly follows. It may by cohesive inflammation and ulceration be evacuated into the stomach, when sickness and vomiting are produced. In like manner it may pass into the colon, or into the pelvis of the kidney, and be voided by the rectum or the bladder. Or the ulcerative process consequent upon pressure may be produced in another direction, and it may go through the diaphragm into the thoracic cavity, giving rise to pleuritis; and in this way in some rare instances the pus has been transferred into the bronchi and coughed up, when it has closely simulated empyema. Mantell not long ago recorded a case in which the matter burrowed into the left lung and was expectorated; and on examination of the organ after death it was discovered to be an empty sac, only containing a very small quantity of dark brick-coloured fluid, which sampled with that expectorated. When the pus is carried into the kidney the symptoms of uræmic poisoning are rendered in greater or less degree apparent. When the secretion seeks an external exit the pointing may be anywhere between the ileum and the costal margin, or it may be between the umbilicus and the lumbar muscles. I have known the matter burrow through the abdominal wall and perfect recovery succeed. Grotanelli and Raikem record instances of spontaneous outcome of the matter through the abdominal parietes.

Reference has already been made to embolic infarction, caused by cardiac disease, and the localized inflammation which such event is liable to institute in the gland. The abscesses which follow are usually not large at the commencement, but they are apt to coalesce, and thus from this cause we sometimes meet with a considerable collection of pus; they give rise to perisplenitis, seldom to general peritonitis, and the capsule covering the subjacent deposition becomes thickened and its peritoneal investment is rendered vascular and opaque. At an earlier period of the formation of these spots a dark and congestive zone surrounds the embolized part, and the process of softening is first discovered in the centre of these cuneiform deposits. When of pyæmic origin or resulting from the secreting surfaces of surgical operations or suppurative wounds, the tendency to soften and assume the conditions of abscess is more marked than when the infarction is from pieces of lymph being carried in the circulation from heart disease. Sometimes hydatids produce abscess in this organ.

SYMPTOMATOLOGY AND DIAGNOSIS.—It has already been stated that the symptoms of suppuration in this organ are uncertain and imperfectly pronounced; just as we know such uncertainty to obtain in hepatic abscess. There may be little or no pain, as is generally the case when the pus is formed centrally, and without the involution of fibrous and serous investments. There may not be either nausea or vomiting, if no irritation be given to the branches of the vagus. Before fluctuation can be absolutely felt there is always room for doubt as to the presence of the suppurative process. When fluid in the viscus is diagnosed, rigors and flushes and copious perspiration are the common objective symptoms. There is not unfrequently a hard dry cough caused by diaphragmatic pressure, and the pulse is usually full, quick and compressible. If, however, the peritoneal coat become inflamed, the pulse, instead of being large, acquires the characteristics of the pulse of peritonitis, by being of less volume and firmer under the finger. Pain in the left shoulder is not uncommon, and this fact was long ago pointed out by Grotanelli. More than half-a-century since he asserted that in splenic abscess there is pain in the left scapula and left shoulder. There is often aching behind the acromion process, and this dull pain may extend to the chest and up behind the neck. The pain in the side may remain for a considerable period stationary; it may increase or it may decline. If the matter do not quickly accumulate, the amount of pain remains the same. When it becomes augmented, this symptom may go on to be excessive. When there is absorption the pain gradually declines. When the matter makes its escape into one of the hollow viscera or externally, the mitigation of the pain is marked and sudden, and it may be at once abolished. The secretion may pass into the cavity of the peritoneum and alarming symptoms are marked and immediate. A great shock is given to the system by this grave and very commonly fatal event, the phenomena of which are readily interpreted. The pulse becomes quick and feeble, the features look pale and sunken, the surface is often bathed in a cold, dewy perspiration, there is a tendency in the extremities to become cool, the decubitus is on the back, with the knees drawn up, and the respiration is thoracic. After the supervention of these alarming conditions the mind is often clear, and it may remain unclouded to the last. With these grave changes the splanchnic nerves become potently impressed, the deep cardiac plexus and the branches given off to that plexus by the pneumogastric become paralysed, and death eventuates from gradual arrestment of the heart's action. Collections of pus sometimes form in the abdominal walls, and such might take place over the splenic region; but in such instances the tumefaction is more superficial, and careful manipulation discovers it to be located in the parietes. In the course of time there is a brawny feel to the integuments beyond the limits of purulent deposit; they feel hard and tight, and the lines of dulness are ill defined. Again certain negative facts come to our help in forming a judgment on the point. In parietal abscess appearing in the place now named, it must be held in mind that the characteristic conditions of splenic disease would be wanting. The clinical history would tell of no endemic cause; there would be no account of intermittent or remittent fever; the peculiar cachexia of miasmatic poisoning would not be apparent, palpation would not discover the dependent edge of the organ, and the patient would be able to lie on the right side. Empyema might burrow and point over the situation of the Spleen, it might seek an exit as I have known it between the crest of the ileum and the costal edge, but the common cardinal and physical signs of pleuritic effusion would be our guide. The tumour of psoas abscess is not so high up as splenic tumour caused by suppuration, and pressure over the psoas muscle is likely to produce bulging of the fluid in the groin. Renal abscess and encysted kidney give indications by testing the urine. Leucocythæmic enlargement of the Spleen importantly differs from splenic abscess. The hardness of the first-named affection, the falling downwards into the pelvic cavity, the absence of rigors, flushes and perspirations, would indicate the true nature of the malady. Malignant growths in this situation would be readily excluded by the calling to mind many negative as well as positive facts whereon we base our conclusions relative to the presence or absence of carcinomatous growths.

PROGNOSIS.—All authors and the best authorities are agreed that abscess of the Spleen, whether coming on from malarious causes or of pyæmic origin, is always fraught with great danger. In many so-called recoveries it was, perhaps, by no means absolutely certain that a suppuration had existed. In some few cases it is possible that absorption may take place, and the patient get well, just as this event, in exceptional instances, may follow purulent formations in the liver. There are various cases of a favourable termination recorded when the matter has by ulcerative absorption found its way into one of the hollow viscera, or when it has burrowed through the diaphragm into the bronchi; but the more frequent result is the catastrophe of the fluid's extravasation into the cavity of the peritoneum. It may, as before remarked, find an outlet through the parietes, but this mode of its escape is very rare, and even when it occurs, death generally ensues from protracted secretion, and the irritating and debilitating effects of a large suppurating cavity. When from pyæmia, it may be said that abscess of the Spleen is almost always fatal.

TREATMENT.—The treatment required in this affection is very much the same as the treatment required when the same kind of pathologic change takes place in the hepatic viscus. Both being solid organs, both contained in the abdominal cavity, and both being influenced by like impressions, it can be easily understood how they are subject to similar morbid processes. If the pain come on suddenly with accelerated pulse and exalted temperature, and the patient can with difficulty take long inspirations, such would infer inflammation of the fibrous and serous coverings. With these symptoms in a case occurring in this country, or in a patient who is a European and who has not been long resident in the tropics, and whose constitution has not been broken down by protracted exposure to the malarial influences, by intemperance, or other debilitating causes, leeches may be applied. Warm fomentations should also be used, and large poultices placed over the part. The latter may be made of bran or linseed-meal, or partly of linseed-meal and partly of bread. They should be placed into a muslin or flannel bag, covered with cotton wool, and then a piece of oiled-silk should be spread over the edges. By this mode the heat and moisture are much longer retained, and thus the necessity for the local application being repeatedly changed is avoided, and consequently the patient is less inconvenienced. When there is symptomatic fever, saline purgatives, diaphoretics, and diuretics should be prescribed. The aperient may be the sulphates of soda or magnesia, Pullna water, the citrate of magnesia, or bitartrate of potash. A dose of the Carlsbad salts in half a tumblerful of soda-water sometimes answers very well. When the patient's strength is much reduced, the preparations of senna, rhubarb, or aloes may be given, and to any of these a small quantity of podophyllin may be added, whereby a bilious evacuation is promoted. In order to maintain an action on the skin, the acetate liquor of ammonia, in combination with the nitrous spirits of ether and camphor julep, may be prescribed. The nitrate of potash and the bitartrate of potash will keep up the function of the kidneys. When there is gastric derangement with flatulent eructations, the alkaline mineral waters are to be commended, particularly those of Vals, Vichy, and Ems. With the decline of the symptomatic fever quinine should be given in full doses, and most especially when the primary disease is of endemic origin. It ought also to be conjoined with sulphuric acid. When there is much pain, harassing cough, as there may be by implication of the diaphragmatic surfaces, some of the preparations of opium may be needed. It is always desirable that the patient's strength should be husbanded by continuous and refreshing sleep. Upon opiates the most dependence can be placed. In some instances the bromide of potassium, or ammonium, or the chloral hydrate, may be sufficient to calm the nervous system and give a good night's rest. It may be repeated here, what has already before been insisted upon, that mercury in any form should be avoided, either as an internal remedy or as an outward application. When the abscess points externally it is generally better to allow nature to effect an opening than to have recourse to the knife. There is then no injury done to the walls, and no shock given to the system. And where the matter is being spontaneously evacuated there should be no squeezing or pressure, and large and soothing cataplasms should be kept constantly applied. Under this plan of treatment the matter will escape in a slower way, but it is a more desirable mode of its evacuation. Harm is generally done by instrumental interference. When an artificial opening is made, air enters the sac and fresh inflammation is liable to be excited. A puncture can hardly be made without hæmorrhage into the splenic substance. It must, however, be conceded that a hard and fast rule on the point in question is not to be contended for, because in certain instances there might be the danger of expectancy. The continuous and unremitting augmentation of the fluid would jeopardise more and more the giving way of the sac, and if the rupture were into the abdominal cavity, that event, as it has repeatedly been observed, means death. If there were a projecting fluctuating tumour, with a pointing inflammatory blush, and it appeared that the integuments were resistive, a small trochar might be introduced, or Bowditch's aspirator might be employed. The canula of the trochar might, if its presence produced no irritation, be for a few days left in, or a pledget of oiled lint be placed in the orifice, and the instrument after an interval introduced. A large and moist poultice should after the operation be applied, and a full dose of morphia or Battley's liquor opii sedativus be immediately given. With regard to food, such articles of diet should be selected as are nourishing and easily digested. Strong beef-tea, which might be thickened with corn-flour, sago, tapioca, or rice; veal and ham broth make an agreeable change; and game and chicken panada are likely to be relished by the patient. Good milk, if the patient can take it, is always desirable, and it often agrees better with lime-water. It can be given in soda-water, and occasionally in champagne it suits very well. Light sherry, claret, burgundy, or marsala are perhaps the best stimulants.

CONGESTION AND HYPERTROPHY OF SPLEEN.

That congestion of this viscus may continue for a protracted period without giving rise to inflammation is unquestionable, and, as before observed, its distensile qualities favour the one and not the other condition. It is referrible to mechanical obstruction, to fluxionary hyperæmia, or to a dyscrasic state of the fluids, though it may be produced by impediment to the circulation of the vena porta or cardiac disease. As a rule, its hypertrophy is not owing to mechanical causes; not unfrequently in heart disease, or cirrhotic and nutmeg-liver, the organ is found smaller than normal. In acute endocarditis, which implies blood disease, splenic hypertrophy is often produced. Wilks and Moxon assert that in ten cases of ulcerative endocarditis its average weight was as much as twenty-five ounces, and these authorities say that in ischæmia, without poisoning of the circulating fluid, and where inflammatory deposits did not exist, the gland was generally rather under than over weight. Its far more common cause, however, is the malarial influence, and those blood changes which miasmatic emanations engender. Wilks and Moxon say its hypertrophy when considerable is due to disease of its texture or of the blood. Between the blood, when contaminated with miasmatic poison, and this viscus there is some special and peculiar relation. Mere pyrexial exaltation of temperature is not the cause of its enlargement. There must be in addition some absolute and sanguineous change. The accession of its hypertrophy may be sudden or slow; if the former, the pain is urgent, and if the latter, there may be no pain. Vascular engorgement is more or less rapid, according to the injury which the constitution has received from the [illegible] a the antecedent attacks of periodic fever which the pa[illegible] varying times undergone. In temperate climates thi[illegible] state is of a passive character, being by no means so bro[illegible] as in tropical regions, where intermittents and remittents [illegible] The hyperæmia may be temporary, and only cognizable dur[illegible] the cold stages of an ague; but if the vascular turgor be continually repeated, the fibrous structures and the proper parenchyma become hypertrophied—there is hyperplasia. Whether the unusual accumulation of blood depends upon increased action of the arteries, or on inability of the veins to remove it, is a question not decided, but the results are the same, stasis being effected enlargement must follow. In aguish districts it is not uncommon for persons to have splenic enlargement for many years, and die from ot[illegible] diseases; and it has been stated that individuals have had hy[illegible]ophy of this organ through life. Haller and Lieutaud mention s[illegible]

Often it must needs be an [illegible] distinction between congestion and a low and an ill-defi[illegible]ammation, knowing as we do by what insensible gradations [illegible] respective conditions pass from one into the other. Baillie says [illegible]ere may be congestion with healthy structure. This, however, wil[illegible] depend much upon the degree of the hyperæmia. Craigie believes its increase of volume to be an incipient morbid state which will progressively terminate in another morbid state. Primarily, simple congestion may eventuate in inflammation, and congestion and hypertrophy stand in relation of cause and effect. An often-repeated afflux of blood not only mechanically distends the organ, but conveys an abnormal amount of those assimilative materials which produce excess of nutrition, and consequently abnormity of the tissues. The effects of such sanguineous afflux are in other ways apparent. Hyperæmia in an organ is succeeded by impairment of its functions, and the sum of such impairment stands in just correlation to the amount of hyperæmia. If then the office of this gland be the elaboration of the formative materials of the blood, the evolution of an albuminous plasma from which the germs of the blood-corpuscles are derived, and, too, according to Kölliker, the dissolution of the old and effete corpuscles, the morbid enlargement of this organ must necessarily have a deleterious reaction upon the entire volume of the sanguineous fluid, and also upon the general powers of the constitution; hence it becomes explicable why congestion and hypertrophy are associated with pallor and sallowness, diminished vital action, and all the debased conditions of anæmia.

SYMPTOMS AND DIAGNOSIS.—When the congestion comes on suddenly the subjective symptoms are more pronounced than by mere dulness, aching, and dragging in the side; there may be in addition absolute pain, which is accompanied by more or less of febrile phenomena. There is a complaint of fulness and uneasiness under the left costal edge, and moderate pressure confers the feeling of soreness and tenderness; there is often some cough and inability to lie on the right side. Aching of the shoulder is another fact not uncommonly mentioned, and not unfrequently the stools are very dark, evidently malænal, when the urine is pale. The digestive and assimilating functions become impaired, the epigastrium is rendered smooth and rounded, and often there are eructations and flatulency. Upon investigation it will generally be discovered that there is an unfavourable state of the constitution. The conditions more distinctive and observable of the malarial influence are in most cases discernible. In the more chronic examples the dull, dead, puffy, facial expression, the pale conjunctiva, blanched lips, gums, and tongue, unmistakably bespeak the anæmic change. Loss of flesh and strength are the common accompaniments, and the tendency to hæmorrhage attests the morbid alteration of the blood, and the want of nutrition in the capillary walls, which such alteration has produced. The skin is often cool, the pulse slow and compressible, and the temperature not more than the normal standard. In cases of long standing in which the organ has become hypertrophous, it often happens that the lymphatic glands are large, more especially the submaxillary, inguinal, and axillary, which is a co-existent pathologic change that can readily be understood from what has already been said in a foregoing part of this article. Niemeyer thus accounts for this symptom of lymphatic enlargement:—"When the cells formed in the intertrabecular spaces of the Spleen, or in the cells of the lymphatic glands, are retained in any way, and are not normally borne along by the blood and lymph, they accumulate in these places; the Spleen or the lymphatic glands, as the case may be, swells up and the blood becomes poor, as the used-up blood-corpuscles are not replaced by others. If the obstruction to the passage of the young cells into the circulation be removed, the growth of the Spleen and the lymphatic glands ceases, and the blood is gradually improved by a sufficient supply of young cells, even if the enlarged Spleen or lymphatic glands do not increase in size." The facts to be observed in diagnosis are very similar to those enumerated under the head of Chronic Splenitis; ordinarily, congestion, as before insisted upon, is very generally the prelude to those more absolute morbid changes which constitute the inflammatory process, and it thus may be a mere arbitrary distinction which defines where the one state terminates and where the other state begins. In such instances the absence of febrile phenomena would be a weighty negative fact, influencing our decision. When the organ acquires increase of size, as the concomitant or sequel of one of the types of continued fever, the state may be regarded as that of mere congestion. The hypertrophous enlargement comes on more insidiously, the hardness is more proclaimed, nor is there so much tenderness to the touch, and investigation will mostly discover the malarial cause, and if such be not the case it will generally be found that there has been a foregoing hepatic disease which has produced organic obstruction to the portal circulation. The general appearance and the constitutional circumstances should always be carefully reviewed in the endeavour to arrive at diagnostic correctness.

TREATMENT.—When the history of the case points to malarial causes, the speedy removal of the patient from endemic influences becomes of course the first recommendation. Quinine and the various preparations of Peruvian bark are also to be sedulously taken. MM. Caron d'Annecy, Balby, and Piorry place the most reliance on cinchona. The removal to a more healthy climate would doubtless often suffice, but the cure would be unquestionably aided by the specific medicine. In the more active examples of congestion, when there is sharp pain, shallowness of breathing, and considerable distress, those means may very properly be had recourse to which have above been given when describing the treatment in chronic splenitis. The secretions and excretions should receive our attention, and the depurating organs be maintained in active function. Cholagogue aperients by freeing the hepatic system have a good indirect effect on the gland. The venous radicles of the portal vein should, by salines, the decoction of aloes, by senna, rhubarb, or scammony, be acted upon. Tonics such as gentian, quassia, calumba, with occasional aloetic and myrrh pills, are effective, and the sulphate of iron, or the tincture of the perchloride of iron, should in some combinations be taken at the same time. Ferruginous medicines with sulphuric acid constitute a most effective means of treatment. The natives of India have from time immemorial employed a nostrum composed of garlic, aloes, and vinegar, and as a topical application the actual cautery. Shulbred's powder, consisting of jalap, rhubarb, calumba, scammony, the bitartrate of potash, and sulphate of iron, has for half a century been a favourite remedy in Bengal. Twining's formula is a compound of jalap, rhubarb, calumba, ginger, bitartrate of potash, sulphate of iron, senna, and mint water. In India the kala-nimuk, or black salt, of the bazaars is another remedy of ancient use. It consists of the muriate of soda, sulphur, muriate of lime, and the black oxide of iron. The decoction of the seed of the carum nigrum with acetic acid or lemon-juice is also an Indian medicine. Martin thinks highly of the saccharated carbonate of iron, the tincture of iron, and the tincture of iodine, and also the iodide of lead administered externally. This physician in addition recommends the iodide of potassium, warm baths, stimulating frictions to the splenic region, and gestation in the open air. Like most of the writers on this disease, who can speak from experience in intertropical countries, he warns against blisters, as they are apt to slough, and lays emphasis on the fact that mercurials are most injurious and destructive. At the Netley Hospital a combination of the phosphates of quinine, iron, and strychnine has been successfully used. The nitro-muriatic acid with bitter infusions is often of decided service. The diluted nitric acid, applied locally, and the nitric acid bath, have by some been regarded as very effective. The tincture of iodine diluted in the proportion of one part of the tincture and six or seven parts of water, painted over a large area of the splenic region and abdominal surface, is sometimes very beneficial. The natural chalybeate waters are often followed by a diminution of the gland. Grotanelli speaks of the practice of percussion over the splenic region as an old method of cure, formerly had recourse to it in Southern Italy. In the more chronic or hypertrophous enlargement the same kind of remedial measures may be pursued. Dry friction in such examples is serviceable. The hepatic secretions should be looked to with much care in these cases. Henry, Andral, and Piorry insist on the good effects of the cold douche. In hot weather cold sponging and sea-bathing assist other means adopted. The diet should receive particular attention. If there should be any symptomatic fever, and high temperature in the earlier of the congestive stages, the food should be mainly farinaceous, and if milk agree with the patient it may be given. Soups, broths, and jellies and light puddings may be allowed, and even where there is no febrile disturbance too liberal a supply of animal food ought not to be permitted. The aerated waters are relished and to be advised as drinks, such as soda, seltzer, potash, and lithia, and the Apollinaris waters. Dry sherry, Marsala, claret, and Burgundy are perhaps the most desirable wines, which the patient can take when stimulants are indicated. Places of residence of greater altitude and drier atmosphere are often followed by marked advantage.

SPLENALGIA.

Splenalgia is a non-inflammatory and suddenly painful affection of the viscus which the older authors termed dolor lateris and splenis dolor. Voight describes in careful detail what he names splenalgia congestionis, but the affection to which he refers is evidently one of those more protracted forms which are occasionally met with of chronic splenitis. Sudden and powerful muscular exertion, by rapidly determining the blood to the internal parts, and there disturbing the equilibrium of the circulation, will give rise to it. The organ, as it has before been observed, being a diverticulum to counterbalance the inconvenience which would otherwise ensue from inequalities in the visceral circulation, its quick distension with the vital fluid renders the tunics tense and consequently painful. In certain hysterical and uterine affections, more especially in attenuated and anæmic females, the gland is sometimes sympathetically affected, the cause being a morbid sensibility of the nerves proper to the part, just as we know under like circumstances these anomalous pains in nervous patients incident to other organs and localities of the system. During the cold stage of an ague, by a rapid afflux of blood, it will become acutely painful, and the pain immediately subside on the supervention of the hot stage. Its adhesion to the diaphragm is not unfrequent in those more especially who have resided in tropical climates and miasmatic districts, and who, during the progress of periodic fever, have had the organ distended and inflamed. This morbid attachment to the diaphragmatic surface has been considered, and not without show of reason, to be the cause of that sudden, and sometimes severe, pain experienced over the splenic region. Young people of both sexes, after violent exercise, will complain of stitch in the left side. Great runners induce this condition.

SYMPTOMS AND DIAGNOSIS.—The affection may be recognised by its sudden accession and its rapid subsidence. The absence of the ordinary febrile phenomena will point to its neuralgic character. The patient takes long sighs; he places his hand involuntarily on his left side; he tries to make a deep inspiration and cannot; his trunk is slightly curved to the left; and there is a disposition to remain in one position. The pulse and temperature are normal, and the manner of its coming on makes no suggestion as to its being inflammatory. It cannot well be mistaken for pleurisy, because pleurisy is ushered in by a different train of symptoms. That disease supervenes more slowly, more progressively; the hard, dry cough, the pulse, heat of skin, and the physical signs distinguish the one from the other. In pleurodynia, which is an affection of the intercostal muscles, or the fibrous fasciæ lining the chest, and mostly a rheumatic affection, and though in the great majority of instances it is in the left side, it can with moderate care be discriminated from splenalgia. In pleurodynia sudden twisting and movement of the thorax aggravate the pain. Digital pressure on the intercostals increases the discomfort; it is more commonly seen in men than in women, and in those whose systems are lowered from some debilitative cause, or who live in damp houses and cold and wet situations.

TREATMENT.—Such agents as are usually employed in neuralgic affections are to be used in splenalgia. In those of nervous and excitable temperaments the restorative and tonic treatment is generally the most to be relied upon. Ferruginous and chalybeate remedies are chiefly indicated. The class of agents usually called nervine tonics, such as the various preparations of iron, zinc, valerian, and ammonia, are to be commended. The natural mineral waters which are ordered in the condition of lowered vitality and general debility are frequently beneficial. Stimulating liniments, friction, change of air, and underflannel clothing, are to be suggested. In such cases as may come on from violent and undue exertion, rest and quiet are the obvious recommendations.

TUMOURS OF SPLEEN.

Tumours of this organ can mostly be distinguished from other tumours by their obliquity from above downwards, by their lying from left to right. They are sometimes very large. Pemberton mentions a Spleen which weighed three pounds. Morgagni saw one of eight pounds and a half. Elliott describes one of eleven pounds. Bree declares this gland may be so augmented in size as to weigh from twenty to thirty pounds. Portal knew it weigh thirty. Lieutaud found it in a woman, who had been long ill, weigh thirty-two pounds. And Twining states that in extreme cases in Bengal this viscus has been discovered so large as to fill up half the cavity of the abdomen. There are two forms of configuration which it usually assumes on its tumefaction, and these are the rounded, or globular, and the oblong. The globular results from disorganisation of the blood, and it is the accompaniment of an acute dyscrasia such as obtains in periodic, malignant, relapsing, and exanthematous fevers. Rokitansky believes this condition to be dependent upon stasis affecting the vascular system of the fundus ventriculi and the deposition of a dark pultaceous mass somewhat resembling the medullary matter found in the typhus mesenteric gland. The oblong is of firmer consistency, the edge being often notched and fissured, and the colour is not so dark, it being of a yellowish or reddish-white appearance. It gives evidence of a more sthenic type of inflammation. There is adhesion of the tunics; fibrinous matter is deposited in the parenchyma, and this deposition, by encroaching on the vascular network, gives rise to pressure, whereby the colouring particles of the blood are absorbed, and hence the lighter hue. This is its configuration in leucocythæmia. Tumours having as their cause suppression of menstrual or hæmorrhoidal discharge, assume the last-named appearance, which is the condition of a slow and gradual degeneration. In order to detect the presence of the organ when tumid, the reader is referred to what has already been said under the head of General Symptomatology.

General Morbid Anatomy.

It may be remarked that in observations made on the appearance of this organ as revealed by dissection there is not unfrequently no little difficulty in arriving at a correct conclusion, because the morbid conditions so gradually merge from one into another that it must needs be but an arbitrary distinction where the attempt is made to nicely define pathological changes. In the inspections made on many other dead parts it is not so, as the cadaveric alterations are more characteristic and cognizable than they are in this viscus. The colour and configuration of the organ vary under the different morbid processes to which it is subject. It is discovered paler or darker than normal, or it may be of blackish-brown colour. In malignant and putrid fevers its colour sometimes approaches to black. It may be rendered rounder or more elongated than normal, and its fine border sometimes loses its sharper shape. And I have seen it much flattened and more spread out than natural. Andral considered that it would confer preciseness and facility in these investigations if our researches were brought to bear on two component parts; that which is contained, the blood, and those fibroid, serous, vascular, and other structures which go to make up the main substance of the gland. It may be said that the rease of size and variation of consistence are the most usual and iking changes which result from its disease. By long and conuous hyperæmia the capsular coverings become thick and dense, nd the trabeculæ in corresponding manner are rendered larger and re numerous. When acute inflammation has obtained, and localized ritonitis has taken place, the serous covering is then found thick, que and unyielding. Sometimes it is white and nodulated. stowe, on microscopically examining a specimen of fibroid degeneraon of the capsule, found transverse sections of the fibrous tissue to hibit interlacing hyaline bundles, which in the situations where lcareous deposit was found spreading, contained in their interstices number of highly refractive granules, most of which disappeared with e evolution of a quantity of gas on the addition of dilute hydrochloric id.[1] This observer also goes on to say that on section being made allel to the surface of the organ, the interlacing fibroid bundles uld not be recognized, though planes of almost structureless tissue re still to be seen more or less covered by refractive particles similar those which were seen in the previous sections lying in the interstices tween the fibres. The under surface of the diaphanous membrane adherent to the fibrous tunic, and externally the organ becomes tached by holding masses and bands of lymph to contiguous viscera, the abdominal parietes, and in some instances to the concave surface the diaphragm. The serous coat may be noticed red and vascular. metimes there are in the adventitious products loculi filled with rum in addition to the serous exudation which is effused into the bdominal cavity. The inflammatory process, as the almost unexptional rule, extends deeper than the capsular investments, and if reful examination be made, it very generally occurs that a greater or ss amount of thickness of inflammatory exudation can be detected in he parenchymatous substance. Indeed, in all examples of acute ritonitis it can hardly be affirmed that the inflammation is absolutely nfined to the serous membrane. Under the inflammatory condition the fibrous parts may take or contain heterologous transformations, they ay become in greater or less degree cartilaginous or even osseous.

SOFTENING OF SPLEEN.

Softening is a pathologic change more frequently discovered in this viscus than any other morbid appearance, and it is much more common than induration. It is sometimes impossible to say whether the softening be from mere blood stasis or inflammation. In some instances of sudden death the organ has been found large and soft when there were no reasons to suppose the existence of any foregoing disease. Softening is the common condition which follows pernicious, periodic, malignant, and relapsing fevers. The specific poisons generative of febrile diseases, by contaminating the entire current of the blood, are followed by local as well as the more obvious general phenomena, and organs and tissues through an impaired nutrition exhibit in varying degrees the more manifest conditions of absolute lesion. Under the influence of these poisons this viscus is particularly prone to the loss of vital cohesion—in other words, to a breaking up of its ultimate structure—and is certainly much more prone to such disintegrative change than any of the other solid abdominal organs. This altered state of the blood gives rise to the want of normal molecular affinity; the pulp becomes, it may be, semi-diffluent, and the capsule holds not a consistent substance, but debased, broken-up, bloody contents, and the contained grumous fluid has been likened to the muddy lees of red wine, or even unto tar. The gland sometimes resembles a dark resistless clot, invested by an attenuated and tender membrane, which, on the most careful handling, breaks, the contents flowing out as a black, inorganic, putrid gore. In the more malignant forms of remittent fever such appearances have been recorded. The first morbid transformation is deepening of colour; it assumes a dark brown, or it may be black. The pulp degenerates into deliquescence, and the trabeculæ are reduced to shreddy fibres. A stream of water poured upon the loose substance readily washes away the pulp, and the trabeculæ remain, giving the appearance of stringy fibres. The various accounts given of the Walcheren fever record marked illustrations of this condition; and the organ was often seen to weigh four or five pounds, and looked like a membranous bag filled with tar. All authors who have carefully described the morbid appearances in typhus, enteric, and relapsing fevers have mentioned alteration of colour and softening as the common characteristics of this organ. In typhus, in well-marked instances of splenic lesion, the parenchyma is rendered so soft and resistless that it readily breaks under the fingers, and it may be converted into a diffluent grumous fluid. It was soft in fifteen out of twenty-two cases examined by Peacock, and in thirteen out of thirty-one examined by Jenner. Murchison says that in this disease it is not unfrequently reduced to a reddish-brown pulp, which runs out when the capsule is divided. In enteric fever Louis found it soft in thirty-four out of forty-six cases; Jenner in four out of fourteen, and Murchison in ten out of twenty-one cases. Andral gives similar testimony. The blood in this type of fever is seen to contain an excess of white cells, sometimes amounting to three or four times more than are found in healthy blood. From extensive personal experience in relapsing fever, I can assert that there is no organ so frequently discovered to have undergone alteration in structure and consistence as this gland, and softening is also uniformly observed. In this type of fever it is rendered larger and softer than in typhus and in the enteric form. In a number of inspections which I many years ago made on the bodies of those who had died of relapsing fever I never saw the viscus healthy. It was sometimes remarkably large and soft, the capsule was not prone to become inflamed, inflammatory products were not found on its surface, but by pressure it was more readily ruptured, the parenchyma easily broke on pressure, the contents were

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[1] Trans. Path. Soc. vol. xviii. pp. 257-8.

place, partial or more general softness is the common accom-
ıent. In anæmia and hydræmia, and what is termed the
ical crasis, the organ may be rendered softer as well as paler
natural.

INDURATION OF SPLEEN.

luration of the gland is seen in very various degrees. It may
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change will depend upon an increase of vital cohesion in the
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nuous hyperæmia is succeeded by hyperplasia, more organizable
rties may be formed in the cells, and in low forms of its
mation lymphic deposits may eventuate in the loculi or amongst
lamentous tissues. In this abnormality of firmness the changes
n nearly all instances accompanied by enlargement and varied
urations. There is, too, much difference in the colour externally,
when sections are made of its parenchyma. Sometimes, as
t remarks, the substance on being cut looks like damson cheese;
in other instances, as in leucocythæmia, it is of a pale dirty
v. Diemerbroeck describes a black induration. On the whole it
be asserted that lymphic deposit is the chief cause and character-
of its more resistive condition. Sometimes it cuts with consider-
ardness, and sometimes I have seen it so friable as to break like
eese. In those exceptional examples in which the cartilaginous
ation is manifest the entire fibrous structures of the organ are
ented in bulk and increased in density. The envelopes then
e this transformation. That condition when the organ is enor-
ly enlarged, and which Bright called *fleshy hardness*, was evidently
ypertrophous condition now regarded as the invariable and dis-
ishing pathologic change in leucocythæmia. The older physicians
eously looked upon induration as identical with scirrhus; and

where there has been renal wasting, or great periodic discharges of blood, it has been found small and shrivelled. Bree says it has been discovered so diminished as to be of the smallest size, the capsule only containing a little of its vascular substance; Thomson declares it may be reduced to the size of a walnut, or even less; and Andral records that he saw it not larger than a walnut. Dr. Church showed a small spleen at the Pathological Society, taken from the body of a woman who died of pleurisy and ascites, which only weighed five drachms and fifteen grains. It may be discovered in a mere rudimentary state; and it has been asserted that the viscus is sometimes wanting.

HÆMORRHAGE INTO AND RUPTURE OF SPLEEN.

Hæmorrhage sometimes occurs. Blood may be extravasated into the parenchyma without solution of the tunics, which occurrence has been by some morbid anatomists named "apoplexy of the spleen," from the like extravasations which are discovered in the brain and in the lungs. And more accurate observers have noticed that occasionally the remains of apoplectic effusions are detected in the presence of circumscribed reddish-brown spots, which are permeated and surrounded by lymphic deposits. These have been regarded as the abiding evidence of antecedent vascular solutions. In very exceptional instances the coats may give way, and blood be effused into the abdominal cavity, when fatal peritonitis would, if the quantity were at all large, eventually follow. The toughness and strength of the fibrous covering provide against this event, and render the accident of its rupture exceedingly rare. In those cases which have been recorded of this catastrophe, there has generally been a foregoing or primary, and mostly a febrile, affection of pernicious or malignant type, which has contaminated the entire circulatory fluids, and in secondary manner injuriously acted upon the muscular and fibrous tissues. Acting as a diverticulum, as it does to the visceral circulation, and thus necessarily being liable to be surcharged with blood, nature has provided this organ with coverings which are stronger and more elastic than the coverings of the other solid viscera; and, I believe, unless the tunics have by foregoing lesion lost their normal resilience, and thus been rendered liable to solution of continuity, that their giving way never occurs, or that such event is infinitely rare. Sometimes the coats of the vessels spontaneously enlarge, and thus under pressure may give way. Traube and Cohnheim related an instance of sudden death from rupture of a series of dilated veins of the Spleen. These veins were so elongated and distended that they were six inches in length, five in breadth, and two in thickness. Rupture is most liable to supervene in the hyperæmia of pernicious fevers. In tropical countries when the viscus from endemic causes is liable to great distension, comparatively slight blows, falls, or compression are sufficient to produce its laceration. The giving way of its coats does not in any case necessarily imply a fatal termination, as instances have been recorded where a cicatrix has shown the former solution of its tunics, and when the patient has lived for a long time afterwards. Such examples have, however, resulted from accident, they were of traumatic origin, and when the splenic pulp did not become extruded from the bursting of the diseased and tender envelopes. Rupture has been known to take place during the hot stage of an ague, and in typhus and in cholera. Babington once examined the body of a patient in whom this viscus had been completely detached, and was found loose in the pelvis; and in that instance most violent sickness had preceded death, and the powerful efforts in vomiting were considered as being the cause. Two peculiar cases of spontaneous rupture are recorded by Rokitansky, and these were in the instances of two workmen, aged respectively forty-eight and nineteen years. Both had leucocythæmic tumefaction of the organ; in both examples several pounds of clotted and fluid blood were found in the abdominal cavity, and in both the gland was five or six times larger than normal. Sir James Simpson records three fatal cases of this kind of rupture, which occurred respectively during the pregnant, parturient, and puerperal states. In one case the viscus became large during pregnancy, and the enlargement always disappeared after delivery. A woman showed symptoms of fatal sinking shortly after labour at the sixth or seventh month, and autopsy showed a laceration of an enlarged spleen, with effusion of blood into the peritoneal sac. In another patient the woman, after making some unusual exertion a week or two after delivery, complained of abdominal pain and a feeling of sinking, and then soon afterwards died, when splenic rupture and effusion of blood were revealed on inspection. The late Dr. Cumming delivered a woman by the forceps, who died in the course of a couple of hours afterwards, and examination showed a similar circumstance to have taken place. According to the accounts of the Russian epidemic of relapsing fever in 1864–5, as given by Fittermann and Küttner, in three instances out of seventy inspections the organ had spontaneously ruptured. Mr. Atkinson of Leeds has published[1] an apt example similar to the above. A lady, thirty-five years of age, was suddenly attacked with sickness and violent vomiting, which was soon accompanied by pain in the left side; these symptoms were succeeded by lividity, cold extremities, and all the phenomena of fatal collapse, and death took place at the end of twenty hours. On inspection it was discovered that there was a large collection of blood in the abdomen, which had evidently proceeded from a rent in the gastro-splenic omentum, and the Spleen was found shrunken, pale, and flabby. Dr. Deville of Harrogate has kindly sent the writer particulars relative to an instance of spontaneous splenic rupture which recently occurred in his practice. The patient was a man of thirty-six years of age, and of temperate habits. He had been labouring under an attack of continued fever, and was progressing towards recovery, and was so far convalescent that he was enabled to dress and sit in his chair. An acute pain suddenly came on in the left hypochondrium, and after this he rapidly passed into a state of collapse, and died. Inspection revealed a fissure parallel with and anterior to the hilum lienis, and some of the splenic pulp had been extruded into the peritoneal cavity. The Spleen was exceedingly friable and broke on removal. Blows, falls, and compression have caused its laceration. Professor Fayrer records the accident of rupture in an Hindoo woman aged thirty-five, who fell from a tamarind tree and broke both her arms, and who died of tetanus sixteen days after the fall. The examination exhibited two ruptures in the posterior edge of the organ, the upper edge being very deep. Charcot delivered a woman in whom the spleen of the fœtus was found ruptured, and this accident it appeared had been preceded by two falls, one which the mother had sustained a month and another a fortnight before her confinement. Mr. Richard Davy narrates the instance of a woman who had been run over, who was admitted into the Westminster Hospital, and who died two hours after admission. The portion of Spleen corresponding to the suspensory ligament was completely crushed off from the remaining five-sixths of the organ, and there were three linear rents in the capsule. It has been remarked that the horse under powerful muscular efforts is liable to rupture the Spleen.

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[1] Brit. Med. Journal, Sept. 26, 1874.

GANGRENE OF SPLEEN.

Gangrene of this organ is exceedingly rare, and the accounts of its occurrence are so scanty that nothing reliable can be said of its symptoms. Murchison, however, says that in gangrene of this viscus after typhoid fever there is very rapid sinking. Baillie mentions having found it in a gangrenous condition. The older authors speak of gangrene of the Spleen, but their inaccurate and often erroneous descriptions of morbid changes throw much doubt upon such assertions, as upon many other of their statements. They were not conversant with many morbid appearances as modern pathologists are conversant with them. Recent observers of the most ample experience have not witnessed more than one or two illustrations of this most uncommon diseased condition. Rokitansky bears testimony to its rarity, and speaks of having only once seen it, when it affected the organ to a considerable extent. Ollivier, Portal, and Morgagni, and their contemporaries, doubtless regarded that black, grumous, broken-up state of the pulp which obtains in periodic, yellow, and malignant fevers as gangrene, which, however, differs greatly from gangrene. Hertz of Greifswald lately gave particulars of an instance of this morbid alteration in a young woman who had been admitted into the Greifswald Infirmary for abdominal disease consequent on the abrupt arrestment of the catamenia. The post-mortem examination gave evidence of pleuro-pneumonia with pleuritic effusion, and the diaphragm was partly gangrenous, and the Spleen almost entirely in that diseased condition.[1] Authenticated instances of splenic gangrene may be regarded, and truly, as among the curiosities of medical literature.

HYDATIDS OF SPLEEN.

Hydatids have occasionally been discovered in this organ, but far less frequently than in the liver. They as rarely affect the Spleen as cancer, and when seen are often in association with hydatids of the liver. They may be within the substance, between the tunics, or be attached externally. They are seldom found in the pulp, and mostly in the gastro-splenic epiploon, or in the cysts constituted of the serous investment. There are no rules for their diagnosis; they sometimes, however, produce a bulging out over the splenic region, in the form of smooth hemispherical protuberances. Duroziez narrates an instance of a splenic hydatid which was voided by the bronchi. A man admitted for hemiplegia spat up a substance resembling boiled white of egg which proved to be the débris of an hydatid cyst. At the autopsy the spleen was found to be hollowed out into a vast cavity which contained thick, dark-coloured fluid and dark-brownish flocculi. Wilde gives a case of hydatid of this organ in a girl of eleven years of age, in whom the tumour reached three-fingers breadth to the right of the linea alba, and from a point in the latter, half way between the umbilicus and the pubis, to another half way between the former and the ensiform cartilage. In *St. Bartholomew's Hospital Reports,* Vol. viii. p. 181, Dr. Wickham Legg gives the case of a man who died after having epileptiform attacks and delirium, and on inspection the Spleen was found small, adherent to the diaphragm, and in the upper part was a putty-like mass, within which was crumpled up, in a manner resembling the plicative æstivation of some flowers, a transparent membrane which showed a laminated structure, and thus evidently proved its hydatid character. Dr. Coley showed at the Pathological Society a hydatid cyst connected with this organ of the size of a cocoa-nut. When thus large they may be fatal by pressure being exercised on neighbouring organs, or by being extended into the peritoneal sac, and thus giving rise to flagrant peritonitis. Some very rare instances are recorded in medical literature, in which the cyst, by adhesive inflammation, had become agglutinated to the abdominal walls, and the contents escaped externally and harmlessly. Andral speaks of *other cysts,* which he describes as small vesicles filled with serous fluid, floating in or attached to the splenic veins; and of a cyst with fibro-serous tunic filled with hairs. Wilks relates the case of a Spleen being found in the body of an old woman, whose kidneys had presented cysts. The organ was of the average size, but at its lower end was a cyst the size of a walnut, and on its surface were smaller ones, all of which contained serum and were lined with a smooth membrane. This physician is of opinion that there was in this instance evidence of distinct cystic disease, and he comments upon the rarity of the affection as occurring in this gland, there being only one other specimen of a similar kind in the museum at Guy's. The remains of hydatid cysts are sometimes associated with surrounding fibroid thickening and cartilaginous hardening, or there may be calcareous infiltration. Bastian discovered a shrivelled hydatid cyst, the walls of which were about a line in diameter, and the microscopic examination of this wall was found to consist of innumerable hyaline concentric layers, some of these being forty times as thick as others; on the surface of the inner layer were traces of granular germinal membrane, and in the centre were fat and proteine granules, with amorphous carbonate and phosphate of lime. Manchartus once beheld a cyst in this gland which contained four pints of fluid. Spillman has recorded a remarkable case of cystic hæmatina of this organ.[1] It was of the size of an infant's head, attached to the inside near the hilus. The interior of the cyst was made up of a number of communicating anfractuosities filled with a yellowish fluid containing cholesterine. The character of the growth was deducible from the following facts, namely, the cavity was lined with a single layer of cells like those which form the epithelial lining of the vessels; in the general wall many points of calcareous deposit existed; the inner surface was divided into numerous and intricate spaces; the fluid contained numerous blood corpuscles and crystals; while neither inosite nor succinic acids, both of which constantly occur in hydatids, was found in the fluid.

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[1] Virchow's Archiv. xl. p. 580.

DEGENERATIONS OF SPLEEN.

The degenerations which occur in this viscus, like the transformations which occur in other organs and tissues, are in their ultimate textural changes and in their progress latent and obscure; they depend upon molecular abnormality or some perversion in the assimilative functions, and some vice in the vital endowment of the part which is doubtless referrible to a more general cause, and it is most probable that the first morbid condition takes place in the blood. It may be that there is an albuminoid conversion whereby oil is generated in the circulation, to the decrease and supplantation of normal nitrogenized materials, and hence they form the fatty decay; and that some analogous fundamental error in the vital fluid disposes to fibrous, calcareous, and various other products. These changes are of slow and chronic nature, and the substitutions which they effect are necessarily followed by the more prominent and cognizable phenomena of impaired function.

Fatty degeneration of this gland is sometimes found in association with that change in the heart, liver, and kidneys. There is then seen an excess of oil molecules in the textural and vascular formations. The parenchymatous substance more readily breaks up under the fingers, and according to Rokitansky the blood contained in the vessels is frequently of pale red colour and is more serous.

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[1] Archives de Physiologie, Aug. 1876.

In the *fibrous degeneration* of this gland there is a fibrinous crasis, and certain thickenings and depositions eventuate; according to Henle, they are produced out of an effused plasma and by the nuclei developing short fibres whose union renders them more complete. Compression and atrophy of adjacent textures will be proportionate to the sum of these formative materials. The septa as well as the splenic capsule enlarge in volume, but in the latter the alteration is the most marked. The trabeculæ are rendered stronger and tougher, and thus the gland possesses greater power of resistance, and the tunics look more opaque. Recently, a man aged sixty-five was admitted into the Tunbridge Wells Infirmary, and he died of a low form of pneumonia. On inspection the house-surgeon, Mr. Cleland Lammiman, discovered the Spleen to exhibit the notable appearance of fibroid transformation. The gland had assumed the renal configuration, and the fibrous covering throughout was of dense, white structure, the eighth of an inch thick, and in the parenchymatous substance were found two hard, unyielding fibroid deposits. Gyot relates the case of a man who in early life had had intermittent fever, and after whose death dissection discovered this organ to be covered with a gelatino-fibrous network, and the microscope showed an abundance of fusiform and stellated cells, and other elements which enter into the cartilaginous structure. This kind of degeneration is more common to this gland in those who have had acute splenic inflammation, and in the aged it is sometimes met with as one of those heterologous changes incident to middle and advanced life.

The next pathologic product of which I shall speak, and one which is sometimes observed in this organ, though not so commonly as in the liver, kidneys and lymphatic glands, is that known as the *lardaceous* or *amyloid* substance. Perhaps in the strictness of technical language it can hardly be called degenerative in the same sense as we employ that term in description of the fatty change, when there is absolute substitution of the sarcolin within the sarcolemma; but this adventitious substance by its presence destroys healthy tissue and supplants it, and therefore the results are so analogous that I now refer to it under the present head. It is of albuminoid nature, and according to Wilks its appearance implies a long-standing and deep-seated cachexia, and a foregoing caries or necrosis of bone which has originated in tuberculosis or syphilis. According to the last-named authority, the substance when incised is semi-transparent, and presents the appearance of wax and lard combined, yet it is not fat, nor wax, nor gelatine, but, as Virchow describes it, the product is a peculiar albuminous compound. According to Frerichs and Murchison it is rarely associated with fatty degeneration in phthisical subjects. Dickinson, founding his opinions on the experiments of Hasse and Lehmann, as well as from clinical observation, attributes the cause of this deposit to a loss of albumen and alkali from the blood; and is consequently of opinion that suppuration and albuminuria, and not phthisis or syphilis, are the main causes, and says it is followed by an excess of fibrin in the blood. There are no symptoms during life whereby this substance can be positively diagnosed; but in certain instances of protracted and suppurative caries, when the liver is large, and at the same time the lines of splenic dulness are exceeded, it may be suspected to exist. When after death it is found in this gland, the deposit has in nearly all cases been discovered also in the liver and kidneys. According to Gairdner the lardaceous or amyloid condition is characterised by an increase of firmness being given to the organ; it is waxy looking and the Malpighian sacculi are transparent and distinct. Aikin, in describing this lesion, says the gland is enlarged, and has a swollen aspect, that it feels of the consistence of wax and lard, and that the Malpighian corpuscles are rendered more distinguishable, being round, colourless, prominent, transparent granules, and that the pulp is greatly diminished in quantity and seems in some to be entirely absent; and this writer also asserts that, under the higher microscopic powers, the ultimate change consists of an alteration in the normal corpuscles of these sacculi, which are converted into masses of colourless, dense, homogeneous, translucent material which exhibits irregular cell forms. Busk and Huxley are of opinion that this pathologic change first commences in the arterial capillaries, that the sacculi which at the first contain normal splenic cells, at the later stage contain granular and lymph corpuscles, which ultimately pass into a waxy substance, and that at length the trabeculæ and pulp undergo a similar morbid conversion. Wilks describes three conditions incident to this viscus which are connected with the lardaceous disease, but says that only one of these ought strictly to bear that name. In the true affection he says that round translucent bodies occupy the place of the Malpighian corpuscles, looking like millet seeds, but never encroaching upon more than half of the splenic pulp; that the adventitious product confers some amount of enlargement and density, and that it is not only deposited in these corpuscles, but surrounds the smaller arteries and the trabeculæ. In the second form, according to this pathologist, the same kind of translucent substance looks as if melted tallow had been poured into the cellular structure in an ill-defined and incidental manner, and he believes it to be of strumous origin. The third description given by this physician is of a whitish-yellow soft material deposited in the gland in irregular masses, and which is connected with enlargement of the lymphatic glands. Some have named this pathological state of the organ *sago spleen.* The application of weak tincture of iodine when brushed over sections containing these bodies produces a brown colour, which is characteristic of the lardaceous substance. An apt illustration of lardaceous spleen is given by Dr. Hilton Fagge[1] in the instance of a man who died of Bright's disease. He had voided about four pints of urine daily, which was of light specific gravity and which contained a large quantity of albumen. Inspection revealed proofs of syphilis; there were small soft nodes beneath the pericardium, and the liver contained whitish-yellow tough gummata with soft mucoid centres. Several of the viscera were lardaceous, the liver yielded a slight and partial reaction with iodine, and the right supra renal capsule was affected with this change in marked degree. The spleen weighed ten and a half ounces, was highly lardaceous, and presented the appearance of sago-spleen. It also showed whitish-yellow patches of irregular form, and all reaching the surface; on section they were more or less wedge-shaped, like infarctions, and gave a reaction with iodine. On section their cut surface was of dull yellow, with some greyish, translucent parts. They were portions of the lardaceous change in a state of fatty degeneration. Wilks and Moxon say this morbid change is not confined to the small blood-vessels, but extends to the lymphoid structure, which composes the parenchyma of the corpuscle. The lardaceous and amyloid matters are always found together in the specimen examined.

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[1] Trans. Path. Soc. vol. xxvii. 1876, p. 325.

Hodgkin was the first to point out the fact of enlargement of the absorbent glands in connection with a peculiar white suet-like deposit in the splenic pulp, and which, although it bears a resemblance to lardaceous disease, cancer and tubercle, is certainly identical with none of those products, and which by its destruction of the surrounding healthy tissues may with some propriety be considered malignant, and this organ is its most usual seat, though it is occasionally found in the liver and other of the solid viscera. In the affection more particularly referred to the lymphatic glands become very large, with great uniformity of structure, and their enlargement precedes, it may be for a long time, the splenic affection, nor is there in them a tendency to suppurate. These white bodies located in this viscus are distinct and well-defined, and the more obvious conditions are splenic enlargement, with anæmia and anasarca. The splenic corpuscles which possess this deposit are, as before noticed, in the blood-making functions closely related with the absorbents, and this disease originating in the lymphatics, it can be well understood would, in organs possessing kindred functions, be exemplified in the Spleen. Payne not long ago published a good illustration of this affection in the case of a boy ten years of age, who during life had presented himself with enlarged Spleen, the organ reaching from the seventh rib to the crest of the ilium vertically, behind into the lumbar regions, and in front almost to the mesial line. On dissection this viscus was discovered to contain numerous tumours of the kind called lymphadenoma and infarctions, and the lymphatic glands were similarly affected throughout the body, some containing caseous matter which caused them to be regarded as of a scrofulous nature.[1] A youth of eighteen was admitted into St. Mary's Hospital, under the care of Dr. Sibson, and died some weeks after admission. The lymphatic glands generally were much enlarged. The Spleen was hard and large. On its surface were several projecting masses, and on section the organ was mottled with white masses. This diseased product on being microscopically examined was composed chiefly of round cells, of spindle-shaped cells, or what appeared to be free nuclei.[1] It has been shown that in these tumours polymorphous cells, fibro-nucleated tissue, and an amorphous albuminoid material may coexist in the same case. Dr. Hilton Fagge showed an enlarged spleen and lymphatic glands at the Pathological Society.[2] A large, heavy woman had been quite well until two years previously. She then began to lose flesh, and abscess formed in the left groin. Inspection showed the glands throughout the whole body to be enlarged, with tendency to the suppurative condition. Disease had commenced in the uterine appendages. The spleen weighed twenty ounces, and in it were a number of large masses the size of a walnut containing a cheesy looking substance. There being some evidence of syphilis it is presumptive that the cause of these morbid changes were referrible to specific origin. Wilks, in speaking of this diseased condition, says its peculiarity is in the glandular system being first affected, and the Spleen afterwards becoming involved; and he remarks that it is possible for the propagation to take place in the course of the lymphatics, and the reason why the splenic corpuscles are affected is their intimate connection with the absorbent system. He also states that the adventitious substance when examined by a microscope shows, when taken from the lymphatic glands, an abundance of cells scarcely distinguishable from the normal secreting bodies with more or less fibrous tissues. This adventitious substance, which pathologists have variously named as lymphoid or adenoid or as lymphadenoma, bears the most resemblance to tubercle, and has doubtless been mistaken for that deposit. These anomalous substances are mostly found in the vicinity of blood-vessels, and are not unfrequently surrounded by a rusty-looking product, which appears like blood which has undergone some morbid change. Sometimes these little spots are very hard, and without any appearance of central disintegration or softening. In the liver the material is much tougher and fibro-nucleated, whilst in the lungs, Spleen, and kidneys it is composed mostly of cells which resemble somewhat those of tubercle.[3]

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[1] Path. Soc. Trans. vol. xxii. p. 278.

SYPHILITIC SPLEEN.

Syphilis affects the Spleen as it affects the other solid abdominal viscera; and since the extent and nature of the ravages of that complaint are now better known to the pathological anatomist than was formerly the case, it is now admitted that no organ or tissue possesses an immunity from this specific affection. The liver is more frequently the seat of this disease than this viscus, and when the Spleen does give evidence of the lesion, the liver as the rule presents a similar pathologic appearance. In some examples there may be partial or more general splenitis, in which a low form of inflammation is exemified, implicating a greater or less area of the fibrous capsule and erous covering, as evinced by opacity, thickening, vascularity, fused products and adhesion to contiguous parts. A debased lymphic udation, or, as it has been called, an albumino-fibroid material, is rmed in the parenchyma of the gland, sometimes towards the centre, n other instances near the surface; and the microscope reveals granular lements in the course of retrograde evolution. When syphilitically fected, the organ is very commonly enlarged; hyperæmia, a conseuence of the morbid change, tends to tumefaction, and infarction stitutes the inflammatory phenomena with effusion and inrease of volume. Those gummata which are met with on secon are roundish or circumscribed patches, and may be singular r in great or less numbers; according to Lancereaux, they are een as whitish or yellow-looking nodosities, being frequently deep seated, and quite characteristic of their specific origin. They reatly resemble the localised infiltrations from this cause in syphilitic iver. Sometimes the cut surface looks red and shining, and the gland at the same time has acquired density, as ascertained on pressure being made by the fingers. Induration is the most usual alteration; but in some instances the viscus is rendered soft and pasty to the feel. One of the best descriptions of the syphilitic state of this gland is that given by Virchow. "Under the influence of a moderate hyperæmia," says he, "some parts of the splenic parenchyma become tumefied; sometimes deposits are formed in one or other of the lobes, sometimes the change extends irregularly throughout the whole organ. The affected parts are hard when cut; they appear darker, drier, and more consistent. Sometimes they are of a blackish-red colour, and resemble hæmorrhagic deposits, and it is even difficult to distinguish them from inflammatory congestions. Later on the redness disappears, especially at the centre; the tissue of the organ, while becoming drier and harder, takes on a paler colour; sometimes, on the contrary, it is of a greyish red. From this moment the augmentation of the conjunctive tissue is evident. At the points where the change takes the form of a deposit there is afterwards seen a retraction, or thickening, and a cicatricial depression, as we have seen in the syphilitic lesions of the liver, the testicle, and the iris. White and thickened in such cases, the fibrous capsule of this gland generally adheres to the diaphragm." In addition to palpable enlargement, the most marked event is perisplenitis, the symptoms of which, however, are generally masked or ill-defined and localized. Dr. Gee says that enlargement of the Spleen in children the subjects of congenital syphilis is about in the relative proportion of one-fourth of the cases, that the size of this organ is some index to the degree of cachexy, and that its increased volume continues two or three years after the more manifest indications of the specific disease have disappeared. In several examples which have latterly been published, in which splenic lesion was traceable to the syphilitic taint, the morbid appearances, as before remarked, very closely resembled that infiltrated and denser condition which has been more frequently given of syphilitic liver. And, as Diday remarks, the more attention which is now being bestowed upon visceral lesions in syphilitic children, will supply a better explanation of deaths which hitherto have been vaguely attributed to debility and the more general effects of the poison.

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[1] Path. Soc. Trans. vol. xix. p. 401.

[2] Nov. 4, 1873.

[3] Guy's Hospital Reports. Third Series. Vol. xi. p. 64.

TUBERCLE OF SPLEEN.

Tubercles are sometimes located in this gland, but almost if not always in connection with tuberculous growths in other organs, and generally in children. In acute tuberculosis Rokitansky says the viscus is swollen and softened, and more like the condition it assumes in the typhoid state. Jenner asserts that it is often the seat of tubercle in children, but only in exceptional cases in the adult. The lungs and mesenteric glands are very commonly at the same time diseased, and the tuberculous matter in the splenic parenchyma is seen in minute and scattered grains, or in isolated groups, and may vary from the size of a millet-seed to that of a pea. A patient was admitted into St. Thomas's Hospital, under the care of Dr. Peacock, who died from general tuberculosis, the meninges, lungs, kidneys, and other parts presenting miliary granulations. The spleen weighed fifteen ounces, and was completely stuffed with caseous nodules the size of large peas, some of which were softening, and their yellowish colour contrasted strikingly with the dark-coloured pulp.[1] The tubercles are often distributed throughout the substance with much regularity, and they may be solid and hard; but in the course of time they soften in the centre and assume a curdled appearance, or look like cheesy substance. Billroth says that in miliary tuberculosis of this gland the parenchyma is rendered dark, hyperæmic, and extremely brittle, that the deposit is in the splenic tissue, and that the veins in the vicinity of the tuberculous matter contain a large number of cells with large nuclei very similar to those found in typhus spleen.

CANCER OF SPLEEN.

Splenic cancer is exceedingly rare, and is seldom if ever discovered alone. Jenner bears testimony to its rarity, and thinks he has never seen it except in conjunction with the same growth in other organs. A specimen was recently exhibited at the Pathological Society of what was supposed to be primary splenic cancer, and which occurred in the practice of Dr. O'Connor. A most careful search had failed to discover malignant growth in any other part of the body. The Committee on Morbid Growths, who microscopically examined the product, pronounced it to be carcinomatous, but, as they observed, there had been effusion into the left pleura, with no obvious conditions preceding that event, and there might have been a small and primary mediastinal tumour, thus lessening the probability of this case being, as had been supposed, an almost unique example of primary splenic cancer. In some cases the Spleen and lymphatic glands have both been cancerous, and some have attributed such coincidences to the fact of functional alliance between those organs. It may be found of the schirrus variety, but it is far more likely to be met with in the encephaloid form, such as we discover in the liver. The authority now quoted illustrates carcinoma of this viscus in the instance of a patient under his care in the hospital. A woman was admitted for schirrus of the breast and rectum, and she presented all the conditions of the cancerous cachexia. A large tumour occupied the left hypochondrium and passed up under the ribs, and it was hard, with a sharp edge, which met the margin of the thorax at a right angle. Its dulness extended up to the level of the nipple, and the lower edge was on a level with the umbilicus. It was situated just beneath the parietes, moved up and down with the movements of respiration, and could be pushed a little from side to side by the hands behind and in front of it. Its surface was irregular, and in the anterior border several distinct nodules could be felt; and the increase of dulness, the hardness and irregular surface, and the nodules were precisely the same in kind as are those which are common in carcinoma of the liver.[1] Sometimes the deposit may be central, and neither the configuration nor the bulk of the organ be materially altered; and it is then seen in rounded deposits. The malignant growth becomes invested with a fibrous sheath, and in some cases the product becomes disintegrated within this covering. Mr. Maurice exhibited at the Pathological Society a case of colloid cancer of the Spleen in a man aged thirty-six, and it was remarkable for the very large size which the organ acquired by the presence of malignant deposit. At the same Society Mr. Durham some time ago showed a specimen of splenic cancer in which the organ weighed twenty-four ounces. Its surface presented small whitish, granular growths in the capsule, and the same bodies extended into the parenchyma, varying in size from great minuteness to the size of a vetch-seed, and the section resembled Virchow's illustration of lympho-sarcoma of this gland. When detected in this organ it has not unfrequently occurred, on minute investigation, that the lumbar glands here show evidence of the cancerous product. *Melanosis* may occur in the splenic substance, as it may occur in nearly all other organs of the body. Though it cannot strictly be considered a morbid product in itself, yet this black matter is secreted from the blood very commonly when there is malignancy, and it is under such circumstances that it is found in this viscus.

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[1] Med. Times and Gaz. vol. ii. 1873, p. 22.

OTHER ANOMALOUS GROWTHS.

Sometimes on free sections being made in this organ, masses of *yellow fibrinous matter* of uniform consistence are discovered, which Bright thought to be the remnants of apoplectic clots, but which more recent pathologists would regard as the remains of extravasations produced by emboli. Moxon examined a Spleen in which there was a circumscribed patch presenting the ordinary condition resulting from embolism, and a plug of pale fibrin filled the arterial branch which went to this part of the organ. Nodules and masses of yellow opaque matter are sometimes found in this viscus associated with some foregoing and general disease in the system. Dr. Douglas Powell related the case of a young woman who died of tubercular phthisis in the Brompton Hospital, illustrative of what is now affirmed.[1] On the surface of this gland were many spots varying from the size of a pea to a large filbert, and on section these nodules were found to be firm, opaque, yellow globular masses imbedded in the splenic parenchyma. The vessels were not blocked, as each mass was invested in a thin fibrous capsule, or on minute examination these depositions were seen to consist of a fine fibrous stroma, the fibres of which were granular, and free fatty granules were also detected. They doubtless resembled the cheesy matter known as a product in scrofulous inflammation, and which sometimes bears a resemblance to those gummatous formations which in more external deposits are noted in subjects who have laboured under the syphilitic cachexia. Balfour and Grainger Stewart not long ago published a case of ascites with enlarged Spleen, in which the splenic vein was atheromatous, and in which there were several true aneurisms. The fibrous tunic has been discovered three or four lines in thickness, and occasionally between the serous and fibrous coverings ossific laminæ have been found. Bright once saw, on section of the organ, two pieces of bony matter imbedded in its substance. Valsalva discovered the coats converted into a bony hardness; and Littré exhibited to the Royal Academy of Sciences in Paris a specimen of this gland, the whole substance of which had become osseous. These last-named changes are found nearly always in old people. Rokitansky says they are often noticed as the ossification of fibroid laminæ, or as the cretified fibrine in the cellulo-fibrous callus. Bampfield narrated a remarkable case of ossification in which from the centre of the viscus arose a spherical bony tumour as large as the head of a fœtus seven months old, and it contained seven ounces of serum, in which were floating bright, micaceous, chalky particles. Calcareous deposits have been noticed as free concretions or phlebolithes in the venous channels of the gland. Morgagni recorded an instance in which a calculus weighed twenty-one drachms; and in the Acta Parisiensa, an example is given in which the internal structure of the Spleen was found bony without any other mark of disease. It has been observed in some cases, in which the organ has been larger, and more especially when chronically enlarged, that *White Patches* have been produced on its surface and most commonly on the convexity. The upward and downward motion of the viscus during the act of respiration in such instances, causes local irritation, and thus gives rise to fibrinous thickening of the

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[1] Brit. Med. Journ. Feb. 13, 1869, p. 138.

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[1] Trans. Path. Soc. vol. xx. p. 366.

le. White patches of the same kind are sometimes seen on the
but more frequently, on account of its greater motion, on the
icles of the heart. Their extent and amount of thickness, of
, depend upon the degree and direction of the friction to which
gan has been subjected. *Cicatrices,* like the old seams of former
ons or ancient scars, are occasionally discovered on the splenic
e after death, and such marks leave no doubt as to their having
produced by previous injuries.

Dislocation and Malformation of Spleen.

location of the Spleen is another and remarkable circumstance,
ich a few instances can be found recorded in medical literature.
splacement may be congenital or caused by disease. In absence
diaphragm, and in rupture of the diaphragm, it has been found
n the thoracic cavity. Haller, in the case of a child, saw it lying
e side of the urinary bladder. Desault beheld it in the right
f the thorax. Riolanus on two occasions witnessed it adherent
e uterus. Ballonius once found it attached to the bladder.
lison discovered it resting on the brim of the pelvis, retaining its
neal and vascular connections, and freely moving in all directions.
s and Moxon saw a spleen, of twenty-four ounces weight, entirely
ated and lying in the pelvis, where it might have been mistaken
varian tumour. Küchenmeister has collected several cases in
this organ has been found displaced. Rokitansky has given
examples. The first of which he speaks was in a young man,
he gland was found upon the right ilium attached to a twisted
le, and the pedicle consisted of the pancreas and splenic vessels.
econd case was in a phthisical woman, in whom it had sunk
into the left iliac region, being joined to a long pedicle and to
mentum. In the third illustration the organ was adherent to
igmoid flexure and small intestine, and it was the size of a goose's
A remarkable case was exhibited to the Société d'Anvers by
urns. A woman, aged forty, was admitted into the hospital,
ently suffering from strangulated left inguinal hernia, and she
tumour of about the size of the fist in the iliac region. Vigorous
nent was resorted to, but without success. On examination after
adhesions were found to have formed between the omentum
the intestines, and flakes of lymph in several positions. The
ur was found to be the spleen held by pseudo-membranous bands
e abdominal wall, and also to the intestines, which were very
dilated beneath it. The splenic vessels formed a large cord
ng to the left hypochondriac region, but the substance of the organ
normal. Elongation of the ligaments is the cause of this dis-
ment. Twisting of the pedicle compresses the nutrient vessels,
atrophy of the viscus results. It has sometimes occurred in
inations after death that this organ has presented a shape and
guration rendering it unlike its natural appearance. It has been

found thick and round, and the anterior edge hardly or not at all defined. Sometimes it is more spread out and thinner than normal; or it may be symmetrical and unnaturally small, and it may show rudimentary lines as if two or more Spleens had coalesced. Several pathologists remark upon its occasionally having been seen fissured and lobulated. Riolanus noticed this fact, and records that he had thus seen it double or threefold, and Barthalonius asserted that he had seen it in five portions.

SUPERNUMERARY SPLEENS.

Sometimes one or two or more separate and distinct Spleens have, on dissection, been discovered. They have been observed small as a sparrow's egg or a filbert, and, on examination, their internal structure has resembled that of the larger organ with which they have been associated. Fallopius saw three Spleens in one subject, and Cheselden, Cruveilhier, Hulke, Bright, and others mention having met with a plurality of this gland. Splenules, which are far more commonly met with, are but portions of the larger Spleen.

GENERAL DIAGNOSIS.

It should be held in remembrance from what has already been said in this article, that splenic diseases in this country are by no means so commonly met with as, from the writings of the older authors, it is evident was formerly the case. The reclamation of waste lands, the better and far more extensive drainage, and an improved system of agriculture have told most favourably in the prevention of intermittents and remittents, and consequently in the diminution of splenic diseases. Still, from time to time the miasmatic affection of this gland comes before almost every practitioner of medicine, and the general rules of observance for the detection of the maladies incident to the viscus may now concisely be recapitulated. That condition of the countenance, and those peculiar and more objective symptoms exemplified in the patient, which are so commonly seen in those labouring under one or other form of this organ's lesion, and which are said to enter into the condition constituting what is termed the splenic cachexia, point significantly to the kind of ailment which obtains. The dull, listless, apathetic expression; the dirtyish yellow-hued colour of the skin; the pale and bloodless eyes; the puffed face; and the exsanguine lips, tongue, and gums, confer striking evidence, and which cannot fail of a correct interpretation. The facts elicited in clinical history generally give etiological particulars which are in close accord with the nature of the symptoms; and more accurate examination and attention to the physical signs add positiveness to diagnostic inference. In the young, and those whose constitution has not been undermined by long residence in tropical climates or aguish districts, the cachectic state may be less pronounced;

all, whenever the functions of this organ are in any way interfered with, the blood-making process cannot be normally carried on, and a greater or less degree of anæmia is produced. In certain of its organic and chronic diseases, more especially when there is manifest enlargement, the diagnosis is not difficult; but in some other forms considerable doubt may be left in the mind of the physician, and even after much painstaking and patient investigation. In acute splenitis, when the tunics and convex part are inflamed, the diaphragm not unfrequently becomes implicated, when there are sharp pains, cough, and dyspnœa; the surface is then hot and dry, and the thermometer indicates elevation of temperature, the pulse runs high, and peritonitis may, and mostly does, in circumscribed manner obtain; but the tenderness on pressure is not diffused, or only in exceptional cases, over the abdomen. The patient cannot lie on his right side; he then reclines on the left, or involuntarily assumes a diagonal position. On palpation there is more or less of tenderness over the splenic area, and often on movement of the trunk from the right to the left side there is acute pain. When there is enlargement of this viscus in periodic fevers, the diaphragm may be so pushed up as to compress the base of the left lung; but percussion over the dull thoracic part does not give that dead sound which, when it obtains, indicates fluid in the lower portion of the chest, and the dull space is not varied from the former cause by the altered position of the trunk. The breath-sound is absent altogether; there is no ægophony; no splash is heard on concussion; the dulness is continuous below the false ribs; and palpation generally discovers the round, smooth, or notched edge of the organ. This disease is distinguished from hepatitis by the absence of jaundice and the abnormal colour of the excretions; in liver complaints the patient lies on or inclining towards the right side, and digital examination and percussion mostly confer a correct knowledge of the enlargement of the liver. When the left line of the liver exceeds its normal boundaries, the dulness extends to the right hypochondrium, and it may be held as a rule that in nearly all cases the right lobe is also diseased. Splenic enlargement is known from pleurisy and pneumonia by the non-existence of those physical signs and more general objective or subjective symptoms which are so indicative of these respective affections. There is not the pleuritic pain at some point of the thoracic wall, the pulse is not so quick and full, nor is the temperature so high, as when that serous membrane is inflamed. The absence of the dusky hue of the countenance, the pneumonic sputa, and the shallow, hurried respiration, which are so characteristic of inflammation of the pulmonary substance, are guiding negative facts. It is distinguishable from pleurodynia by the seat of pain and unaffected circulation, more sudden supervention and transient character of that complaint. It differs from nephritis, because in kidney lesion the pain is localised more in the lumbar region, and renal mischief can with certainty be detected by the ordinary tests employed in examining the urine. It may be known from encysted and movable kidney by the dulness being continuously traced up under the thorax and not towards the spine; and, too, by the less mobility of the mass than there is in these alterations now named in the kidney. It does not agree with ovarian tumour, from a general review of the more prominent circumstances associated with ovarian disease. In ovarian tumour there is resonance, in the earlier stages especially, between the upper edge of the tumour and the false ribs, and the enlargement is lower in the abdomen and traceable into the pelvis. It varies from impacted colon, because in the last-named affection the percussion dulness can be followed transversely and in coincidence with the colonic course; there is then often a configuration which to the touch varies from day to day, and a large injection will mostly settle the question. In aneurism of the aorta the pulsation and bruit, and not unfrequently the greater or less degree of paralysis in the legs, tell with much distinctness of that grave disease. In cancerous growth of the omentum, the tumour is flat and hard, and widely spread over the abdominal cavity, and generally there is great and lancinating pain. In carcinomatous enlargement of the cardiac end of the stomach there is generally much sickness, with sharp stabbing pains and sour eructations. In splenic disease there is, as a rule, a history of marsh fever, which denotes the true nature of the malady.

The indications of suppuration are flushes and rigors, throbbing, enlargement, wasting, and declension of strength, a dull aching rather than acute pain in the left side, and without the symptoms of empyema, phthisis, liver disease, suppurative nephritis, lumbar abscess, or ulceration of the bowels. In mere congestion the pulse is usually slow and large, nor is there any marked degree of pyrexia. In leucocythæmic hypertrophy there may be no history of malaria, and the organ extends downwards and forwards rather than transversely; sometimes this kind of splenic tumour half fills the abdominal cavity; and in no other lesion of the organ does it assume such enormous dimensions. Blood taken from a patient under such circumstances and examined by the microscope shows the inordinate number of white cells, which render it different from mere anæmic blood, and which so markedly characterise that affection. In anæmia there is deficiency of red corpuscles, and in chlorotic females the heart is feeble and the liver generally engorged. In the disease now known as anæmia lymphatica, the blood-globules are pinkish, in diminished quantity, and the lymphatic glands are large. The chief and more obvious diagnostic appearances of splenic disease in children are the marked indifference which they exemplify to things around them, the neglect of, and little inclination for, childish amusements, the blanched conjunctivæ, the sickly, pale, waxen complexion, and the loss of flesh and strength. The increased splenic dull area is often distinct, and the abdomen is full and round, and the bowels are irregular. On the examination of the blood there is deficiency of fibrine and red corpuscles. Some writers have said the face of the child often presents a creole shade, which is not the ochery colour of icterus, nor yet the discoloration of chlorosis, but a shade quite special, which, in no very felicitous phraseology, has been called bluish icterus. For more precise details relative to diagnostic differences, and those special symptoms whereby the various lesions of this viscus can be recognised, the reader may recur to what has already been said under the head of Symptomatology.

General Prognosis.

In forming a prognosis relative to the diseases to which this organ is liable, many and varied circumstances should be taken into account, and the diagnostic conclusions will very greatly determine the opinion to be formed as to future eventualities in the course of its respective maladies and the ultimate result. The clinical history and various antecedent occurrences must in all instances necessarily modify the decision at which the physician arrives, and without a knowledge of such foregoing facts no right conception of the issue can be entertained. The probabilities of recovery will depend much upon the kind of complication, the vital powers of the patient, the longer or shorter duration of the affection, and the causes by which it has been produced. When co-existent with other diseases, the nature and degree of severity of such diseases should be duly regarded and the influence which they are likely to have upon the lesion of this viscus. In sthenic types of splenitis or those of unbroken constitution, and when it is the accompaniment of periodic fever, the prognosis is favourable. In pernicious remittents and in adynamic fevers, when there is great contamination of the blood, when the organ has become gradually large, and seems little amenable to treatment, and when there is hepatic, cardiac, or renal complication, with ascites or anasarca, the prognosis is unfavourable. In marked and chronic enlargement, and when the gland is soft and resistless to the touch, and when it comes down below the costal edge and it is preternaturally hard, such changes cannot otherwise be contemplated than as being of grave import. Such instances as are accompanied by hæmorrhagic discharges, more especially by epistaxis and hæmatemesis, generally indicate a serious lesion of the organ; or when there is a persistent diarrhœa with occasional melæna in patients much emaciated, and particularly those in whom these symptoms are in association with general dropsy, there are well-grounded reasons for fear as to the ultimate issue. Again, cutaneous maculæ, vibices, and dark purpureal stainings are very unwelcome appearances. When there are decisive evidences of splenic suppuration, such condition is most ominous, as death generally follows. In simple congestion produced by marsh fever, the primary cause being removed, and a proper course of treatment pursued, recovery, as the rule, succeeds; or if the enlargement is evidently traceable to hepatic obstruction brought on by some functional or temporary occurrence, the gland may fully regain its normal size and natural functions. In leucocythæmic hypertrophy the cases mostly go on to a mortal termination, and the prognosis is then most unfavourable. In cases which are believed to be those of cysts, hydatids, and other anomalous growths encroaching upon the parenchymatous substance, the opinion cannot be otherwise than dubious and full of apprehension. In children and young people in whom has been induced splenic disease from malarial poison, change of district or climate gives great hope of restoration to health.

CONCLUSION.

In conclusion, it may be remarked that miasmatic enlargement and other morbid states of this viscus, as it has already been stated, are amenable to remedial measures, but certain conditions of its chronic hypertrophy cannot be influenced by medicine. Under a due consciousness of this fact the extirpation of the gland has been practised. It is recorded by Gustav Simon[1] that in the case of a young Neapolitan it was removed by Zaccarelli in 1549. A medical friend of my own, who had long resided in India, told me that the operation had occasionally from remote times been had recourse to in that country, but the want of success had led to its discontinuance. It was done by Quittenbaum of Rostock, in 1826; by Kückler of Darmstadt, in 1855; and in November, 1865, it was performed by Spencer Wells. The arguments which have been advanced in favour of the reintroduction of this terrible measure are that its removal from the lower animals has apparently been followed by no ill effects in the system; and that it has been removed in the human subject by accident, as narrated by Morgagni, when the individual subsequently lived five years; and, finally, that it may be atrophied to an exceedingly small size without life being sacrificed or the health materially injured. Such considerations are likely to weigh much in these days when the abdominal cavity can, as it is so constantly shown, be freely opened with the greatest success. The patient operated on by Wells was a married woman, who had had children and was thirty-four years of age, and who was considered to be dying of splenic disease, such being her only ailment. In October, 1865, the viscus extended as high as the seventh rib, and it reached down so low into the pelvis that it could be felt through the walls of the vagina; and the line of union between it and the left lobe of the liver could not by palpation or percussion be determined, nor were there as accompaniments either dropsical tendency or glandular enlargement. The gland rapidly increasing and the patient losing ground, Sir William Jenner was called in consultation, when he detected a soft anæmic murmur over the cardiac region; and on microscopical examination excess of white blood-globules was discovered. Death being inevitable if the case were left alone, the operation was decided upon, and it was terminated in thirty-five minutes under chloroform. She recovered from the shock, but died of pyæmia on the seventh day. The cadaveric inspection revealed effusion into the pericardium and both pleural cavities, but no peritonitis beyond the vicinity of the wound, and no hæmorrhage. The organ weighed six pounds four ounces, avoirdupois, and it measured eleven inches in length, eight in breadth, and between three and four in thickness. Kœberlé lately excised the Spleen in Spain, but the patient died from hæmorrhage. Its ablation not long ago was successfully effected by Péan. Mr. Bryant excised it in June, 1866, the patient being a young man of only twenty years of age, who for a year and a half had laboured under its leucocythæmic enlargement. The tumour filled nearly the whole of the left side of the abdomen. The operation was easily performed and the tumour readily withdrawn from the cavity, but the patient survived only three hours, the cause of death being hæmorrhage into the abdomen, not, as it seemed, from the pedicle, but from a smaller vessel lacerated during the process of separation of the adhesion. The last-named surgeon a second time removed it in a leucocythæmic woman, aged forty, and in this as in the preceding instance hæmorrhage caused the fatal result, the patient bleeding to death in fifteen minutes. Splenotomy is now very generally regarded as unwarrantable. For more ample details on this particular question the reader is referred to the more recent works on surgery.

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[1] Gustav Simon, Die Extirpation der Milz am Menschen. Giessen, 1857.

SPLENIC LEUCOCYTHÆMIA.

By W. R. Gowers, M.D.

Synonyms.—Leukæmia, Splenæmia, Virchow; Leukæmia lienalis; Splenopathia leucocythæmica, Magnus Huss; Diathèse lymphogène à forme leucémique, Jaccoud.

Definition.—Leucocythæmia[1] (λευκὸς, *white*, κύτος, *cell*, αἷμα, *blood*) is a chronic disease characterised by a considerable and permanent increase in the number of the white corpuscles of the blood, the tint of which is in consequence paler than usual. The change in the blood is in most cases accompanied with enlargement of the spleen, alone or associated with enlargement of the lymphatic glands; sometimes also with a change in the medulla of bones; very rarely with the change in the lymphatic glands alone, and still more rarely in the medulla of bone alone. A slight increase in the number of white corpuscles exists in many diseases, and has been termed "leucocytosis." It is not usual to regard a case as one of leucocythæmia, unless the proportion of white corpuscles is considerable and permanent.

Nature and Relations.—The change in the blood which constitutes the characteristic feature of leucocythæmia, a considerable and permanent increase in the white corpuscles, is associated in all, or almost all, cases with two other conditions, a diminution of the red corpuscles of the blood, and an overgrowth of adenoid tissue in certain organs of the body. These two conditions characterise also other forms of disease not accompanied by an excess of pale corpuscles in the blood. The diseases characterised by these conditions (the adenoid overgrowth with anæmia, or leucocythæmia, or both) have points of identity fundamental and numerous.

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[1] The name (leucocythæmia) proposed by Bennett, is preferred to that of Virchow (leukæmia) on the ground that, as pointed out by Parkes, the blood is never white, and rarely approximately so, that the term white blood had before been applied to a totally different condition, and that the term of Bennett describes what is universally taken as the diagnostic sign, the microscopical condition of the blood. This may be marked when there is little or no change in its appearance to distinguish it from simple anæmia. Moreover, the general adoption of the word leucocyte to designate the cells which are in excess (proposed by Robin in 1855,) gives peculiar aptness to the term leucocythæmia. No doubt the blood, as Virchow objects, never consists solely of leucocytes; but it is never white, as it should be if termed leukæmia. The word leucocythæmia is at least as free from objection on the score of not being literally exact, as most other designations of disease. Probably its orthography and pronunciation might be with convenience assimilated to other terms by spelling it leucocytæmia.

The tissue known, since the description given of it by His, as "adenoid," consists of lymphoid corpuscles embedded in the meshes of a "retiform" stroma. Normally it is located chiefly in four situations—in the spleen, the lymphatic glands, the medulla of bone, and in certain glands of the alimentary canal (follicles at the base of the tongue, tonsils, solitary glands of the stomach, solitary and Peyerian glands of the intestine). An overgrowth of this tissue, with more or less change in its characters, may occur in one or several of these situations, and in each may be associated with simple anæmia or with leucocytal excess. In most instances there may be also a new growth of adenoid tissue in situations in which normally no tissue of this structure can be recognised. From the various combinations of these pathological elements there result diseases of widely different aspect. Between some of these the distinctions are considerable, but between all the members of the series intermediate forms exist, so as to justify their classification into one group of "lymphatic" diseases, and to render their separation a matter of some difficulty.

This difficulty is increased by the circumstance that we are ignorant of most of the conditions on which the pathological differences depend. We are ignorant of the precise relation between the growth of the adenoid tissue and the blood state. We know little of the origin of the symptom which has usually been taken as the guide to classification, the presence of an excess of white corpuscles in the blood. As a striking and remarkable characteristic, it was naturally seized as the most salient, if not the most important, feature of the morbid states in which it occurs, justifying the separation of these from other conditions. But a wider observation has shown that each of the pathological conditions mentioned above may or may not be accompanied by this excess, without, so far as our observation goes, any necessary pathological difference in the recognisable characters of the tissue changes. Having regard therefore to this similarity of the conditions in which leucocythæmia does and does not occur, and in our ignorance of the precise conditions on which it depends, it seems unscientific to separate altogether the morbid conditions in which it occurs from those, otherwise apparently the same, in which it does not occur. To do so is to elevate to the rank of a disease that which, according to our present means of observation, is only a symptom—comparable to that of albuminuria.[1] It seems more consistent with the principles on which the classification of disease is based, to group the cases according to their initial and predominant pathological lesion, as far as it can be determined, assuming, as their connecting character, the common facts of progressive anæmia and adenoid overgrowth.[2]

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[1] Barth, Vigla, &c.; Bull. de la Soc. Méd. des Hôp. 1856, p. 60. Magnus Huss expressed a similar opinion, Archives Générales de Méd., 1857.

[2] Jaccoud has done this so completely as to describe the whole class of affections, from splenic leucocythæmia to mycosis fungoides of the skin, as one disease, "Diathèse lymphogène."

It will be found, however, that the two methods of classification lead to results which are not wholly at variance. The majority of cases of lymphatic disease present primary lesions in one of two seats of adenoid tissue, the spleen or the lymphatic glands. Out of one hundred cases taken without selection (as recorded within certain years) the spleen was the first and chief seat of disease in fifty-seven cases, and the glands in forty-three. Of the former there was a considerable excess of white corpuscles in the blood in fifty-four; and in many the excess was very great. It was absent in three cases only. (It is probable that this proportion of cases of splenic anæmia is too small and arises from such cases being less remarkable, and less commonly reported.) In the cases of primary glandular enlargement there was an increase of the white corpuscles in thirteen only, and in none of them was the excess very great. In thirty there was no excess. Thus leucocythæmia is common and often extreme in the progressive anæmia which accompanies splenic enlargement, and is comparatively rare and then slight or moderate, in cases of progressive anæmia accompanying primary glandular enlargement. The two forms of affection present, moreover, some difference in their etiological conditions, in their course, and in their termination. The division into these two classes is therefore adopted in the following articles; and as simple anæmia with splenic enlargement has been considered in the article on Diseases of the Spleen, the two forms of disease are described as Splenic Leucocythæmia, and Hodgkin's Disease, or Lymphadenosis. The rare forms with primary intestinal and medullary growth are described incidentally.

HISTORY.—Each of the elements of which leucocythæmia consists was recognised by the older medical authorities. On the one hand, cases of enlarged spleen, accompanied by the special symptoms which we now know to be common in leucocythæmia, were recorded by many authors from Hippocrates downwards; and on the other hand, examples of a peculiar alteration in the colour of the blood, suggesting the admixture with it of pus, were recorded by Bichat and others in the early part of this century. The association of the change in the aspect of the blood and the enlargement of the
/ ✓ spleen (to 10 lbs.) was described by Velpeau in 1827.[1] In 1839 a case of splenic tumour came under the care of Barth at the Hôtel-Dieu, in which, post-mortem, the blood was noted to be semi-purulent. It was examined microscopically by Donné, who found it to contain "more than one-half white or mucous globules." This observation, certainly the earliest microscopical recognition of the disease, was not published until 1855,[2] but the fact of the occurrence of this change
2 ✓ in the blood was distinctly mentioned by Donné in 1844,[3] and referred by him to imperfect transformation of white into red globules. In

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[1] Revue Méd. 1827, ii. p. 218.

[2] Société Médicale des Hôpitaux, Nov. 28, 1855. Vidal, Gaz. Hebd. 1856, 201.

[3] Cours de Microscopie, 1844. "J'ai plusieurs fois rencontré dans le sang de malades, des proportions considérables de globules ayant tous les caractères des globules de pus, et

1845 two cases of this disease were published together,[1] the one by Dr. Craigie, the other by Dr. Hughes Bennett. Dr. Craigie's case was observed in 1841, and the blood, examined after death by Dr. John Reid, was found to contain "large numbers of pus and lymph cells." The spleen was greatly enlarged, weighing 115½ ozs., and the liver was enlarged to 99 ozs. There was also a conspicuous enlargement of the solitary glands of the ileum, and white spots were scattered through both kidneys. The abnormal elements of the blood, regarded as pus-cells,[2] were referred by Dr. Craigie to the enlargement of the spleen. He ingeniously argued from the structure of the organ that in chronic inflammation, pus-corpuscles would pass into the blood as soon as they were formed, instead of accumulating in abscesses. In the case published by Dr. Bennett the change in the blood was also found by him after death. He also regarded the corpuscles as pus, discussing and rejecting the hypothesis that they were white blood-corpuscles. He attributed them to suppuration of the blood itself rather than to the change in the spleen. 3

A month later Virchow published an analogous case in which also the blood change was only found after death.[3] He pointed out the peculiar nature of the affection, and proposed for it the name of "leukæmia," recognising the identity of the abnormal elements of the blood with white blood-corpuscles. 4

In these observations the state of the blood had been recognised only after death. In the beginning of January, 1846, a case occurred at St. George's Hospital in which the liver and spleen were greatly enlarged, and in which Dr. H. W. Fuller found, on examining the blood both during life and after death, that there was a large proportion of abnormal colourless globules. He described the case at the Medical and Chirurgical Society in June, 1846. In August of the same year Dr. Walshe, at University College Hospital, demonstrated to his class that in the blood of a patient with enlargement of the spleen the "colourless corpuscles were as numerous as the coloured discs." In Germany the disease was first recognised during life by Vogel in 5

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que j'aurais infailliblement considérés comme tels, si je n'avais pas connu d'une part, la grande analogie de structure et de forme des globules purulents avec les globules blancs du sang, et de l'autre si la nature de la maladie et l'autopsie n'avaient pas éloigné toute idée de pus circulant avec le sang," p. 197. "Un homme, dans la force de l'âge, était atteint d'une artérite qui affectait spécialement les vaisseaux des membres inférieurs; les deux jambes étaient le siége d'ecchymoses, de phlyctènes gangreneuses, etc. Le sang de ce malade présentait une telle quantité de globules blancs, qu'en raison même de la nature de son affection j'étais porté à croire que le sang était réellement mêlé de pus; mais, en définitive, il ne me fut pas possible de constater une difference tranchée entre ces globules et les globules blancs. * * * Je suis plus porté à croire aujourd'hui que l'excès des globules blancs tient plutôt au défaut de transformation de ces globules en globules rouges, à une sorte d'arrêt dans l'evolution du sang, qu'à la presence de globules d'une nature etrangère comme ceux du pus," (p. 135). He also (p. 99) attributed to the spleen the function of converting the white into red gobules, as Hewson had done in the preceding century.

[1] Edin. Med. Journal, October, 1845.

[2] According to a statement made after Dr. Craigie's death by Dr. Hughes Bennett, who claimed the discovery of the disease, the significance of the change in the blood was not appreciated, or indeed its occurrence remembered, by Dr. Craigie, until it was found in the second case by Dr. Bennett.

[3] Forieps Notizen, Nov. 1845.

1848.[1] In August 1846, Virchow republished the cases recorded,[2] and brought forward many arguments to prove that the cells were really white blood-corpuscles and not pus-cells, as Craigie and Bennett had maintained. He followed this by a series of very able articles on the physiology and pathology of the blood-corpuscles in relation to the new disease, of which he described several fresh cases, and pointed out the implication of the glands in the morbid process.[3] In 1850 a well-observed case was published by Dr. Parkes.[4] In 1851 Dr Hughes Bennett published a number of fresh cases and facts, and discussed the pathology of the disease in a series of papers,[5] which were in 1852 collected as a treatise on the disease.[6] He adopted the opinion that the cells were really colourless blood-corpuscles, and proposed for the affection the name "leucocythæmia." Since then a very large number of articles and theses on the disease have been published, of which the more important are those of Vidal[7] (collection of cases), Magnus Huss,[8] Ehrlich[9] (collection of a hundred cases of splenic and glandular disease), Ranvier[10] (researches on the changes in organs), Neumann[11] (discovery of the changes in the medulla), and Mosler (many cases since 1864, and a systematic treatise on the disease[12] in 1872). Systematic accounts have been given also by Isambert, and by Jaccoud and Labadie-Lagrave in the French Dictionaries now publishing, and to these articles copious bibliographies are appended. Other very important contributions to the subject have been made by Schreiber, Hewson, Wilks, Charcot, Friedreich, Böttcher, Müller, Ponfick, Scherer, Salkowsky, Kottmann, Waldeyer, and others, and will be referred to in connection with the subjects of which they treat.[13]

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[1] Virchow's Archiv, Bd. 3, 1849, p. 170. Several writers state that Vogel described a case, diagnosed during life, in Canstatt's Jahresbericht for 1846. I have been unable to find any ground for the assertion in Vogel's report on diseases of the blood in the volume for that year. Vogel, in his article in Virchow's "Handbuch der spec. Path. u. Therapie," Bd. 1, p. 393, gives 1849 as the date of the publication of his earliest observation during life.

[2] Med. Zeitung, 1846; Nos. 34 and 36.

[3] Archiv. f. path. Anat. 1847, 1849, 1853, 1854, and Gesammt. Abhand. 1856.

[4] Med. Times, 1850.

[5] Edin. Med. Journ. 1851.

[6] Leucocythæmia, or White Cell-Blood in Relation to the Physiology and Pathology of the Lymphatic Glandular System. Edin., 1852.

[7] Gaz. Hebd. 1856; a series of papers republished as a separate work.

[8] Archives Générales de Médecine, 1857, p. 291.

[9] Ueber Leukämie, Dissert. Inaug. Dorpat, 1862.

[10] With Ollivier, Société de Biologie, 1866 and 1867; Archives de Physiologie, 1869; Histologie Pathologique, 1873.

[11] Archiv der Heilkunde, vol. xi. 1869, p. 870, and in vols. xiii. and xv.

[12] Die Pathologie und Therapie der Leukämie. Berlin, 1872.

[13] In the above sketch of the history of leucocythæmia I have avoided any discussion of the much-debated question of the priority of the discovery. Opinion on this point will depend on the view held as to what constitutes a discovery. It is certain that the disease was first fully observed by Donné and Barth, and first fully described by Craigie and Bennett. If their interpretation was less in harmony with received views than that appended to the independent, and almost simultaneous, description of the disease by Virchow, it must be remembered that modern views as to the nature of pus corpuscles render their explanation, especially that of Craigie, less divergent from the current pathology than it has seemed to be.

VARIETIES.—Leucocythæmia has been divided into varieties according to the seat of the primary or chief adenoid overgrowth with which it has been associated. Thus we have "splenic," or "lienal," and "lymphatic" forms. To these it has been proposed to add a "myelogenic" form, in which the marrow of bone is diseased, and an "intestinal" form, in which the chief overgrowth is in the follicular glands of the intestine. "Amygdalean" and "cutaneous" forms have even been proposed, in which the chief lymphatic overgrowth takes place in the tonsils and as cutaneous tumours. But it is doubtful whether the latter are, as primary changes, ever associated with a large excess of pale corpuscles in the blood, and their distinction as forms of leucocythæmia seems superfluous. The existence of a simple myelogenic form unaccompanied with changes in the spleen or glands, and yet comparable in its course and consequences to the splenic form, cannot be considered as at present established. The primary lymphatic form is described in these articles as one of the varieties of Hodgkin's Disease.

Compound words have been employed to designate the intermediate forms which combine the characters of the supposed varieties. Thus, in the "spleno-lymphatic" form, an initial splenic enlargement is associated with glandular swellings, and in the "spleno-myelogenic" form, with changes in the marrow. The cases again in which the glandular enlargement is distinctly secondary to the splenic tumour, must be distinguished from those in which there occurs a simultaneous enlargement of the glands and spleen, cases which combine the characters of lymphadenoma and splenic leucocythæmia, and may be termed a "lymphatico-splenic," or, more accurately perhaps, a "lymphadeno-splenic" form.

ETIOLOGY.—Very little is known of the causes, immediate or remote, to which splenic leucocythæmia is due. In a minority of the cases some morbid condition or influence can be discovered to which the disease is sequential in time, and of which it may be, in some degree, the result. In the majority of cases no such antecedent can be detected; the disease comes on in the midst of good or fair health. The etiological conditions which can be traced or suspected stand in a relation to the disease which is too uncertain to allow them to be classed as "predisposing" and "exciting." It will be convenient to consider them together, beginning with the more remote.

Race has apparently little or no influence on the occurrence of the disease. It occurs in many countries, and in people of very various nationality and colour. The effect of this influence must of course be kept distinct from the effect of endemic conditions.

Heredity has not yet been definitely proved to exercise any influence. In one or two cases there has been a family history of splenic disease in one or more members of the family. It was so in a case narrated by Biermer,[1] in which a child died at the age of four and a half years of leucocythæmia, and a sister suffered from an enlarged spleen and simple anæmia, and other members of the family died between one and two years of age. A still more remarkable instance is recorded by Casati.[1] Two brothers suffered from progressive increase in the liver and spleen. The father suffered from enlargement of the abdomen, one of his brothers had leucocythæmia, another an enlarged liver, and a cousin an enlarged spleen. The father's mother also suffered from an enormous enlargement of the spleen. We are not told to what extent the family was exposed to malarial influences. In some other instances there has been a history of family ill-health without there being any history of affections allied to leucocythæmia. Thus in a case described by Dr. McCall Anderson, the patient's father had died of some disease of the bowels, the mother of some chest affection, a sister had suffered from a growth, and other members of the family had died young.

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[1] Virchow's Archiv, vol. 20.

Sex.—The influence of sex on the occurrence of leucocythæmia is very distinct. It is twice as frequent in men as in women. Of 153 cases of the primarily splenic form, 100 occurred in males and 53 in females. The mixed lymphadeno-splenic form presents a similar relation to sex. Out of 14 cases 9 occurred in males and 5 in females.

Age.—The influence of age on the occurrence of the disease is also well marked. It may occur at all ages. It has been observed in very young children, even in those at the breast. On the other hand, several cases are on record in which the patients were over seventy years of age. The following table represents the proportion of deaths in 154 cases in the several decades of life. On account of the difficulty, often impossibility, of fixing the date of the commencement of the affection, it has been thought better to compare the ages at which death occurred:—

	Males.	Females.	Total.
Under 10 years	2	2	4
10 to 20 "	11	4	15
20 " 30 "	22	10	32
30 " 40 "	35	11	46
40 " 50 "	16	17	33
50 " 60 "	5	8	13
60 " 70 "	5	1	6
70 " 80 "	5	0	5
Total	101	53	154

It thus appears that more than two-thirds of the cases occur between twenty and fifty years (111 out of 154), and that nearly one-third of the total number of cases occur between thirty and forty years of age. The sexes present, however, somewhat different relations to age. Three-quarters of the male cases occur between twenty and fifty, and one-third occur between thirty and forty. Between forty and fifty, the deaths are much fewer than between thirty and forty. In women, the deaths in the child-bearing period of life, from twenty to forty, are equal in each decade, but in the next ten years, that in which the climacteric occurs, they are more frequent than in any other period, and the number of deaths is almost the same in each sex. In the next decade, that between fifty and sixty, there is a great diminution in the number of deaths, and the fall is greater for men than for women; so that during this decade, and during this only, the cases for women are much more numerous than those in men. During the last three decades of life the numbers of males are equal, which, considering the diminution in the number living, indicates an increasing proclivity in the male sex with advancing years. In women, on the other hand, the disease is almost unknown after sixty years of age, only one case being on record.

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[1] Schmidt's Jahrbücher, vol. 155, p. 327; from the Rivista Clinica, 1872.

Condition of Life.—A comparison of a number of cases shows that the disease occurs with nearly equal frequency in the single and in the married. The female sex affords some exception to this in the relation of the disease to child-bearing, but the effect of pregnancy is not sufficient to render the general rule inaccurate.

It is said that leucocythæmia is much more frequent in the lower than the upper classes; in those who are exposed to privation than in those who live in ease and luxury. The accuracy of the statement is open to grave doubt. A large proportion of the published cases of leucocythæmia have occurred in the poor; but this is chiefly due to the accidental facilities for observation and publication afforded by hospital treatment. The experience of some physicians in large private practice is that the disease is at least as common in the middle and upper middle classes as in hospital patients.

Depressing Physical Conditions.—Intemperance has appeared in a few instances to have predisposed to the occurrence of the affection. These cases are so few, however, only three in 150, that it may be doubted whether it has any real influence.

Inanition is supposed to favour the development of the disease. It is certain that many of the cases presented very ill health for a long period before the occurrence of the symptoms of the disease, and that in some of these the ill health was clearly traceable to extraneous influences, such as deficient food.[1] In several cases depressing emotion appeared to be a predisposing or exciting cause of the affection, and to be followed immediately by symptoms of a cachectic condition which ushered in the pronounced affection. In one case, for instance, severe mental distress, lasting several months, was followed by dyspepsia, and this by a cachexia, in which the abdominal pains, indicative of commencing enlargement of the spleen, supervened.[2] In other instances mental distress has co-existed with some other occasional cause of leucocythæmia, and some share in the effect may be ascribed to both causes. So, in one case, much mental distress followed childbirth, and was succeeded in a little time by great anæmia, and a splenic tumour was soon afterwards discovered.[1]

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[1] Simon, Thèse, Obs. iii.

[2] Charcot and Robin, Gaz. Méd. de Paris, 1853, p. 430.

Over-exertion is said to be an occasional predisposing cause, but its influence is very rarely to be distinctly traced.

Sexual Processes in women appear to have a distinct influence in the production of the malady. In one case the disease was ascribed to an exposure to cold during menstruation. In a considerable number of cases its onset appeared related to pregnancy or parturition; of fifty cases occurring in women such a relation was traceable in eight. The same influence is illustrated by the frequency of the affection during the climacteric decade. It is frequent during the whole of the child-bearing period, thirty out of the fifty cases occurring between twenty and forty-five years of age; but this has less significance since it is most common in men also during the same period. During the climacteric decade, however, nearly a third of the whole number of cases in women occur, and the numbers are more nearly equal than at any other period, while after the climacteric period is over, the disease, in women, is almost unknown. In a few cases the symptoms have come on during pregnancy.[2] More frequently they have succeeded parturition; the weakness after labour has continued as chronic bad health, and in a few months a splenic tumour has been apparent.[3] In one case recorded by Dr. R. Paterson[4] the progress of the disease after delivery was very acute.

Previous Disease has appeared, in a considerable number of cases, to be related to the occurrence of leucocythæmia, either from the frequency with which it has preceded, at some period, the development of the affection, or from the immediate relation between certain severe illnesses and the occurrence of the hæmic change.

Of these the first place in importance must be given to intermittent fever. Most statistics have assigned a very subordinate position to intermittent fever as a cause of leucocythæmia, although they suggested its occasional influence. But on this point the statistics were somewhat misleading, because, in all, except the small collections of Bennett and Vidal, cases are included of primary disease of the lymphatic glands, in which (as will be shown in the next article) a relation to intermittent is very rarely traced. For the larger statistics, those of Ehrlich have been taken as the basis; and a considerable proportion of Ehrlich's cases are useless for the comparison, because many of the cases collected are too briefly narrated to be of value, and because he included so many cases of lymphadenoma.[5] The cases of splenic leucocythæmia which I have collected show that intermittent fever has, at some period, preceded the development of leucocythæmia in a considerable proportion of the cases. Out of 150 cases there was a distinct history of intermittent in no less than thirty, or one-fifth. But in nine other cases, the patients, although they had never suffered from intermittent, had lived in malarial districts, in which ague was common; and in these a malarial influence may reasonably be assumed to have been at work. This will give thirty-nine cases in which such an influence was to be traced, or more than one-quarter of the total number.

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[1] Lissauer, Berl. kl. Wochensch. 1868, p. 40.

[2] Bruzelius, Hygeia, 1872, and Virchow's Jahresbericht. 1873, ii. 299; Feltz, Gaz. Méd. de Strasbourg, 1864.

[3] Barth, Soc. Méd. des Hôp. Nov. 1855; Bennett, case xi. case vi., Leudet, Gaz. Méd. de Paris, 1853; Lissauer Berl. kl. Wochenschrift, 1868; Mattei, Gaz. Hebd. 1858, p. 609.

[4] Edin. Med. Journ. June, 1870.

[5] Since Ehrlich's figures have been so extensively quoted and employed, it is as well to state that this rejection of his statistics is based on an examination of the details

The interval between the occurrence of the intermittent and the development of leucocythæmia varied greatly. In some instances the attack of ague occurred so long before, and was of so slight a character, that doubt might reasonably be felt of its relation to the subsequent disease, if it were not that every gradation can be traced between these cases and those in which severe ague immediately preceded the manifestation of the disease. Of twenty-one cases in which the interval between the last attack and the first manifestation of the disease was definitely ascertained, it was found to present the following variation in years:

30 years	1 case.	7 years	1 case.
25 ,, ...	2 cases.	5 ,, ...	1 ,,
22 ,, ...	1 case.	4 ,, ...	1 ,,
17 ,, ...	1 ,,	3 ,, ...	3 cases.
16 ,, ...	1 ,,	1 year	2 ,,
12 ,, ...	2 cases.	8 months	1 case.
10 ,, ...	1 case.	3 ,, ...	1 ,,
8 ,, ...	1 ,,		

The duration of the intermittent also varied. In several cases it was only a few weeks; in others repeated attacks occurred during many years, in one during twenty years. No relation can be traced between the duration of the intermittent and the length of the interval before the occurrence of the symptoms of leucocythæmia. It is remarkable, however, that in some of the cases in which the longest time elapsed before the occurrence of the leucocythæmic affection, the attacks of intermittent, although distinct, were brief. In one case, for instance, a patient suffered from an attack of ague of six weeks duration twenty-two years before the first symptoms of leucocythæmia. In the latter cases and in those in which there had been residence in an ague district, but no distinct atttack of intermittent, the malaria must have led to constitutional or splenic changes which ultimately eventuated in the blood disease. Where an interval had elapsed between the two diseases the ague in some cases had left a distinct and persistent enlargement of the spleen.

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of ninety-eight of his cases (two being inaccessible to me) and the conclusion reached was that of those examined, in twenty-six there was no evidence of any excess of pale corpuscles in the blood, and in some of these it is distinctly stated that there was no excess. Many were cases of primary glandular disease, with simple anæmia.

In a few cases some other acute disease preceded the onset of the leucocythæmia, either immediately, or at an interval during which the patient remained in ill health. In two cases the ill health preceding the splenic change was ascribed to an attack of typhoid fever,[1] one and three years before. In two other cases it was referred to acute rheumatism three and five years previously[2]; in other cases to small-pox[3] and pneumonia.[4] In the latter case, however, there was a history of intermittent.

In four cases there was a history of syphilis, but its etiological relation to the leucocythæmia is by no means clear. In one case, however, the patient had a chancre only three years previously, and in the interval had many secondary symptoms.[5]

Injury.—In four cases the first symptoms were ascribed to a blow on the splenic region. In two cases the blow was upon a splenic tumour. In one case a sprain was supposed to be the exciting cause. An instance in which the disease was ascribed with some probability to a traumatic cause is related by Ponfick.[6] A man received a kick from a horse on the left side, and a "stitch" in the side prevented him from working for some time. After an interval of freedom of some months, the pain recurred, and again ceased, to return with weakness and some shortness of breath; and later, a year after the blow, a splenic tumour was found, and leucocythæmia, which progressed to death. Post mortem, the enlarged spleen was found universally adherent to the adjacent organs by very dense and firm tracts of tissue, and the adhesion to the diaphragm was continuous with a mass of very hard fibrous tissue which infiltrated the adjacent part of the diaphragm.

PATHOLOGICAL ANATOMY.—*BLOOD.*—The characteristic changes in the blood in leucocythæmia may be observed during life in a drop drawn from the finger, or, after death, in the heart and vessels. The colour of the blood is changed; it is paler than normal, and often presents an undue opacity, which contrasts with the clear rose tint of the blood in simple anæmia. When the increase in the number of white corpuscles is very great this change in appearance may be such that the colour is greyish red, and it may resemble a mixture of pus and blood. When the change is slight, the coagulation of the blood drawn into a vessel may not be affected: when more considerable, between the ordinary white and red portions of the clot is a layer of softer consistence, composed entirely of white corpuscles, and the red clot is paler and more opaque than usual, and contains many white corpuscles. When the blood-change is extreme, coagulation is imperfect, and a soft grumous mass results, chocolate-brown in tint, often containing white or yellowish points—an appearance which has been likened to that of the softened spleen. The purulent appearance of the blood may be very marked in some cases, as in a case related by Virchow, in which, when the ventricle was opened, an assistant exclaimed, "There is an abscess!" In rare cases clots have been found of a slimy consistence and semitranslucent appearance, resembling certain nasal polypi. If the fibrin is separated from the blood by "whipping," and the defibrinated blood allowed to stand, it separates, as Donné first pointed out, into three layers, the red corpuscles sink rapidly to the bottom, and constitute a lowest red layer; above this is a pale layer consisting of the white corpuscles, and above this is the layer formed by the liquor sanguinis, from which the corpuscles have sunk.

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[1] Bamberger, Würzb. Verhand. 1856; Bouillaud and Duroziez, Gaz. des Hôp. 1858.

[2] Greenhow, Lancet, 1876, ii. 859; Schlepern, Virchow's Jahresb. für 1873, ii. 298.

[3] Patchett, Lancet, 1872.

[4] Biesiadecki, Stricker's Med. Jahrbuch, 1876.

[5] Mosler, Berl. kl. Wochenschrift, 1864.

[6] Virchow's Archiv, 1876, vol. 67.

Corpuscles.—In all cases of splenic cachexia the absolute number of corpuscles is lessened. Whether there is or is not leucocythæmia, there is always "anæmia." This diminution in the red corpuscles was long ago insisted upon by Virchow as an integral part of leucocythæmia. The diminution bears no necessary relation to the proportion of white corpuscles. Extreme anæmia may be seen in cases of splenic enlargement when there is no leucocythæmia, and so a considerable deficiency of red may co-exist with a moderate or slight excess of white corpuscles. Nevertheless, when an increase in the white corpuscles exists, there is always a considerable deficiency of red, and when there is a very great excess of white there is a correspondingly great deficiency of red; and so by the method of subsidence it is always found that as the layer of pale corpuscles increases the layer of the red diminishes. The number of pale corpuscles is never sufficient to compensate for the deficiency in the red, and so the leucocythæmia is related to an absolute diminution in all the corpuscles of the blood, both red and white. The proportion between the two kinds of corpuscles varies between wide limits. A slight relative increase in the white corpuscles is common, as will be mentioned in the section on pathology, in many diseases besides splenic cachexia, but in them it is usually transient or trifling. In other diseases attended with enlargement of the spleen, more or less closely allied to leucocythæmia, there may be a slight increase in the white corpuscles in the blood. To distinguish leucocythæmia from these various conditions of slighter blood change, Magnus Huss proposed that only those cases should be considered as leucocythæmia in which the proportion of white to red corpuscles exceeds one to twenty. It is hardly necessary to say that this line corresponds to no natural division. Cases of commencing leucocythæmia have been observed in which a splenic tumour was present, and in which ultimately an enormous excess of white corpuscles was present, but in which, when first observed, the proportion of white to red was far less than one to twenty. If this line be adopted, therefore, it must be with the reservation that cases of splenic cachexia are not to be sharply separated from cases of pronounced leucocythæmia, merely because the proportion of white to red corpuscles is less than one to twenty. When the disease has made some progress the proportion of white to red is usually much greater than this; the proportion of one to eight or ten is common, and one to three or four almost as common (Fig. 1). Frequently a still greater proportion is observed, one to two, two to three, and sometimes equal numbers of white and red, and in rare cases an actual excess of white. There often appears to be an excess of white, on account of the closer approximation and smaller size of the red corpuscles, when by counting, the latter are found to be far more numerous. But cases are on record in which careful and accurate methods of observation proved the white to be the more numerous. An instance of this is a case mentioned by Sörensen in which the proportion of white to red was ascertained, by counting after Malassez's method, to be as 68 to 47.[1]

It is not easy to ascertain the frequency with which the several degrees of excess are met with. In a large proportion of the cases which are recorded no attempt has been made to ascertain accurately the proportion of the white to the red. Observers have been content to record the fact of the occurrence of an "excess of white corpuscles," or that the white were "increased in number." It is doubtful also how much weight is to be attached to the vague statement that the "white are equal to the red," since an apparent equality may be found on counting to be really a great disparity. Taking, however, the statements as made, in seventy-three cases the proportion has been defintiely affirmed, and the following table represents the frequency with which the several degrees of excess were met with:—

1 white to	20 red	in 3 cases.
1 ,, ,,	12 ,,	,, 1 case.
1 ,, ,,	10 ,,	,, 1 ,,
1 ,, ,,	7 ,,	,, 3 cases.
1 ,, ,,	6 ,,	,, 1 case.
1 ,, ,,	5 ,,	,, 7 cases.

1 white to	4 red	in 4 cases.
1 ,, ,,	3 ,,	,, 9 ,,
1 ,, ,,	2 ,,	,, 12 ,,
2 ,, ,,	3 ,,	,, 3 ,,
White and red equal		,, 21 ,,
More white than red		,, 8 ,,

It thus appears that of seventy-three cases of splenic leucocythæmia in sixty-four (or 87 per cent.) the proportion of white to red was greater than one to six, and that in fifty-three cases (or 72 per cent.) the proportion was greater than one to four; while in twenty-nine (or about 40 per cent) the white corpuscles are said to have been as numerous as the red, or more numerous.

In this table only cases of primary splenic enlargement are included. In cases in which the lymphatic glands were first enlarged, the excess is very rarely so considerable. In some cases a primary glandular enlargement has been associated with a change in the spleen similar to that in primary splenic leucocythæmia, and a great excess of white corpuscles has been observed in the blood (lymphadeno-splenic rm). In the case recorded by Vogel,[1] for instance, glandular enlargement preceded for above two years any splenic symptoms, and the blood presented a proportion of one white to two or three red. But the spleen was very large, and the seat of simple hypertrophy. Several similar cases, all accompanied with a great increase in the splenic pulp, are on record.[2]

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[1] Virchow's Jahresbericht for 1876, i. 257.

There are great difficulties in estimating the relative numbers of white and red in various parts of the vascular system, and hence there is considerable discrepancy in the conclusions of different observers. In one point all agree, that the proportion is greater on the right than on the left side of the heart. Uhle, for example, found the proportion in the right heart 1 to 1·5, when that of the body generally was 1 to 3·5. Most observers have found the proportion greater in the splenic vein than elsewhere. When equal in numbers in the splenic vein, the proportion in the portal vein was found by Uhle to be only 1 to 2 or 2 to 3. De Pury found a proportion of 1 to 19 in the splenic vein, when that in the jugular vein was only 1 to 40. Schmuziger, however, found in one case a smaller proportion in the splenic than in the portal vein. His results gave—

Splenic vein 1 white to 4·1 red.	Right heart 1 white to 2·8 red.
Portal vein 1 white to 3·4 red.	Left heart 1 white to 3·7 red.

The absolute diminution in the number of corpuscles may be as pronounced as is the relative increase of white. It is only of late that more convenient instruments for numeration have enabled the diminution to be determined. The earlier method, that of Welcker, by which the diminution in the colouring power of the blood was taken as the guide to the number of red corpuscles, afforded evidence of the fact of the reduction, although not of its amount, since the method can only be relied on when the corpuscles are of uniform size and possess the normal amount of colouring matter. If the hæmoglobin is diminished in the corpuscles, only a considerable diminution in the colouring power of the blood can be taken as evidence of a diminution in number of the corpuscles. By this method, however, Welcker determined that in one case, in which the white were to the red as 1 to 11, the number of red corpuscles was lessened to just one-half the normal, 2,500,000 per cubic millimeter instead of 5,000,000. In another case, in which the proportion was as 1 to 3, the number of corpuscles was only 2,000,000. More exact observations have lately been made by the method of dilution.[3] In one case of pure splenic leucocythæmia, in which there was one white to five red, the total number of corpuscles was found by

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[1] Virchow's Archiv, Bd. iii. 1851.

[2] Wolffhügel, Wurzb. Inaug. Abhand., Carlsruhe, 1871; Schmidt's Jahrbuch, vol. 155, p. 230; Rousseau, Thèse, 1874, Case 6; Grisolle and Hémey, Gaz. des Hôpitaux, 1864, p. 161; Schmuziger, Arch. der Heilkunde, 1876, p. 275.

[3] The principle of this method, and the details of the modification of it employed by the writer, are described in the section on Diagnosis.

Sörensen[1] to be 2,160,000, while in another case of spleno-lymphatic leucocythæmia, that already mentioned as presenting an absolute excess of white corpuscles, there were only 1,150,000 corpuscles per cubic millimeter (470,000 red to 680,000 white). This is a diminution of the red to one-tenth of the normal and an increase of the white to fifty times the normal, the total number of corpuscles in the blood being rather more than a fifth of the normal.

The white corpuscles are usually, in splenic leucocythæmia, of the normal size, sometimes smaller than normal, occasionally larger. Frequently corpuscles both larger and smaller than normal may be observed in the same case. The larger ones may be two or three times the average size. Usually only one nucleus can be distinguished without the assistance of reagents, and often no distinct nucleus is to be seen. The action of acetic acid brings into view sometimes one nucleus, but more frequently two, three, or four nuclei, or a single nucleus, which presents a horseshoe shape (Fig. 2). The substance of the corpuscle is usually finely granular, sometimes contains a clear "vacuole," and not unfrequently presents evidence of fatty degeneration; as was noticed first by Bennett.[2] The fat is in the form of strongly refracting granules or minute globules, scattered through the protoplasm, or aggregated in the neighbourhood of the nucleus. Commonly, the more numerous the white corpuscles the larger is the proportion which present fatty degeneration. Jäderholm found that in a case in which the white were to the red as 1 to 7, five per cent. of the white corpuscles presented distinct fatty degeneration, while in another case in which the proportion was as 1 to 3½, fifteen per cent. of the white were degenerated. In some cases no degeneration has been seen, although carefully looked for.

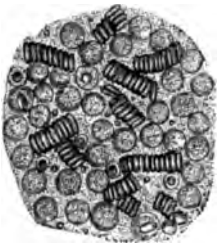

FIG. 1.—Blood in Leucocythæmia. Proportion of white to red as 1 to 3. (After Bennett.)

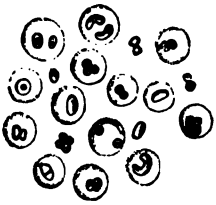

FIG. 2.—Blood in Leucocythæmia, appearance of white corpuscles when treated with acetic acid. (After Bennett.)

The corpuscles of smaller size are most abundant in cases in which the glands are affected, as Virchow pointed out in 1847. Bennett also noted that these smaller corpuscles resemble those found abundantly in the enlarged lymphatic glands. In some cases the

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[1] Loc. cit.

[2] Leucocythæmia, p. 23; Drummond, ibid. p. 43.

een the red and the white cells.[4] Klebs found them in the blood
dead body, and Neumann found them abundantly in the normal
w, and very abundantly in the marrow in leucocythæmia. He
lso seen them in the blood of leucocythæmia during life, and also
e medulla, in which, however, some other observers have failed
d them.

mical Characters.—The reaction of the blood in leucocythæmia
een stated to be acid. Scherer[5] found that the filtrate from the
from the dead body (treated with boiling water) was acid, and Pet-
fer and Voit[6] also found that the serum was acid after dialysis
gh parchment, the acidity being due to a volatile acid, for the
s quickly recovered the blue colour on exposure to the air.
uziger also found the blood acid after death. Scherer thought
the acidity was characteristic of leucocythæmia, and ascribed it
e presence of formic, acetic, and lactic acids. Gorup-Besanez,
ver, found that blood which escaped during life was alkaline.[7]
difficulty of understanding that the chemical processes of the
can be carried on with acid blood makes it probable that the
condition observed after death was the result of post-mortem
mposition.

e specific gravity of the blood is lessened. In health the specific
ity is between 1,052 and 1,057. In leucocythæmia it varies
1,036 to 1,049, the mean being 1,042. The diminution in
ific gravity depends, of course, on a diminution in the amount of
s, and on a corresponding increase in the amount of water con-
d in the blood. The proportion of water in health is 790 parts
1000. In leucocythæmia it is usually more than this. In a case
ained by Scherer, the water amounted to 791 parts only, but in
cases it has varied from 815 to 880, the mean being 838 parts.

e fibrin of the blood is usually increased. In health it does
exceed 2½ parts per thousand. In leucocythæmia it is rarely

sionally to 5, 6, and 7 parts per thousand. The latter proportion was met with in one case by Parkes. The average of ten observations recorded by Bennett is 4·8. Some doubt has been expressed regarding these conclusions on the amount of fibrin present in the clot, since many white corpuscles may be entangled in the clot. It may be pointed out, however, that the mode in which fibrin is now known to be formed, by the union of the fibrino-plastin of the corpuscles and the fibrinogen of the serum, renders its increase in this disease an intelligible fact. Isambert and Robin[1] observed that the fibrin has a peculiar granular character. It does not form long elastic filaments on whipping, but falls to the bottom of the vessel in fragments, in which the fibrillar structure is visible only with the microscope. The same condition had been noticed by Bennett.[2]

The fat in the blood was found by Robertson and Isambert to be distinctly increased; in one case to 7·2 per thousand parts.

The amount of iron is diminished. The normal amount has been very variously stated from ·4 (Becquerel) to ·9 (Jarish) parts per thousand. Strecker[3] found, however, only ·34 of oxide of iron, and Scherer ·29 parts of pure iron per thousand.[4] In the blood analysed by Strecker, the white corpuscles were stated to be one-half or one-third as numerous as the coloured ones. In a case in which the corpuscles were as 1 to 3 or 4 (post mortem), the iron was found by Schmuziger to amount to ·297 parts per thousand.

The albumen in leucocytic blood is said to be diminished, but the statement rests on one analysis only, and very few attempts have been made to ascertain its amount. It was found by Bécquerel[5] to amount to 75 parts per thousand of serum, the other constituents being 907 parts of water, and extractive saline and fatty matter 16 parts.

Various substances have been found in the blood in this disease, which are not present in normal blood, or in the blood of persons with simple anæmia (Sée), at least in quantities which can be detected. Some of these have also been found in the spleen.

Reichardt[6] has described a substance in the blood analogous to that found by Thiere to result from the action of potash on albumin and vitellin, and which was termed albukalin. The formula for it is $C_8H_6N_2O_6 + H_2O$, nearly the same as glycocoll.

Mucin, or a substance closely allied to it, has been found by Scherer, Reichardt, Schmuziger, and others.

A body closely analogous to glutin, has been found by several observers (Mosler, Salkowski,[7] Gorup-Besanez). It differs from glutin in having no action in polarising light, and also in not yielding glycocoll when boiled with sulphuric acid. Salomon failed to find glutin itself.[8]

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[1] L'Union Méd. 1867, 307.

[2] Loc. cit., p. 11.

[3] In Vogel's Case, 1851.

[4] Würzb. Verhand. 1851.

[5] Gaz. des Hôp. 1856.

[6] Jenaische Zeitschrift für Med. 1870.

[7] Virchow's Archiv, Bd. 50.

[8] Reichert's Archiv, 1876, Heft vi. p. 762.

Hypoxanthin, which is found in the spleen in leucocythæmia, was found in the blood by Scherer, and his observation has been abundantly, though not invariably, confirmed.[1] Andrae[2] and Ossikovsky found traces of xanthin, but Gorup-Besanez sought for traces of xanthin without success. Kreatin was found by Ossikovsky.

Leucin has been said to be present, but Gorup-Besanez failed to find it. Tyrosin was found by Folwarczny.

Uric acid was found in the blood by Scherer and Mosler, but could not be detected by Parkes or by Folwarczny.[3]

Acetic, lactic, and formic acids are said to have been found in the blood; and the occurrence of the two latter, which are found also in the spleen, has been well confirmed. They have been found in the blood during life,[4] as well as in the dead body. Acetic acid has been found only after death.

An organic substance containing phosphorus was found in the blood after death by Salkowski, and was conjectured to be a glycerin-phosphoric acid.

Crystals.—Peculiar crystals have been found in the tissues of the subjects of this disease after death. The first published observation of their occurrence was in a case recorded by Charcot and Robin in 1853,[5] the microscopical observation having been made by Robin. Zenker[6] has lately stated that he observed them in three cases before Robin's observation was published. In 1855 they were found in the blood, in all parts, by Mr. Jas. Wallace of Glasgow.[7] They were found in the blood and liver, and carefully studied, by Charcot and Vulpian in 1860.[8] They are elongated octohedra (Fig. 3), colourless, very minute, in average length about ·016 mm., and ·005 mm. in width. Some have been observed larger than this, ·04 mm. in length, and ·008 mm. in width.[9] They always present similar chemical characters, being insoluble in cold water, alcohol, ether, or glycerine, soluble in hot water, and in most acids and alkalies. Their nature is uncertain: it has been conjectured that they are composed of a crystallisable organic substance, an albuminate (Charcot) or a mucin-like substance (Salkowski). The crystals have only been found after death, and hence it is assumed that the substance of which they consist is held in solution during life, and only crystallises out after death. Their appearance, post mortem, may be due partly to the lowered tem-

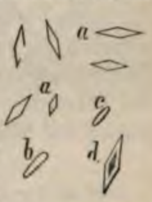

FIG. 3. — Blood crystals (after Zenker). *a*, *d*, regular octohedra; *b c*, with the angles rounded.

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[1] Hypoxanthin has been found by Mosler, Reichardt, Salkowski, Ossikovsky, Gorup-Besanez, and Salomon. It was not found by Andrae or Folwarczny.

[2] Deut. Zeitschrift für prakt. Med. 1875.

[3] Zeitschrift der Wiener Aerzte, 1858, No. 32.

[4] Both by Folwarczny and Körner; formic only by Gorup-Besanez and Reichardt.

[5] Comptes Rendues de la Soc. de Biologie, vol. v. 1853, p. 44.

[6] Deut. Archiv, für klin. Med. vol. xviii. 1876, p. 125.

[7] Glasgow Med. Journal, April, 1855.

[8] Gazette Hebd. 1860, p. 756.

[9] Charcot and Vulpian, Neumann.

perature of the body. Charcot and Vulpian found that they disappeared if the blood was warmed to 60° C. or 70° C., and re-formed a few hours after cooling. The number of crystals is said to increase the longer the time that has elapsed after death. They are not found in healthy blood, and hence it is assumed that either the substance is absent, or that it is held in some combination which prevents it from crystallizing. They have, however, been found in healthy marrow by Neumann.

In leucocythæmia they are found most constantly in the spleen; they have been found in the blood frequently; also in the glands, the marrow, the liver, the spinal cord, and the thymus gland (Desnos). They are most abundant in the spleen, and their occurrence in the blood may be associated with the presence in it of some of the peculiar constituents of the splenic pulp. In the blood they have been seen within the white blood corpuscles, and hence the substance has been supposed to be contained in these corpuscles. The circumstance, however, may also be due to the habit of the white corpuscles of taking small particles into their interior.

They are not peculiar to leucocythæmia; they have been found in the portal blood in a case of simple anæmia, and also in the expectoration in cases of bronchial asthma (Charcot, Friedreich, Leyden). Their presence in sputum has been accounted for by supposing that the substance is contained within the white blood corpuscles, and passes out with them.

ORGANS—Spleen.—Enlargement of the spleen is characteristic of splenic leucocythæmia. The degree of enlargement is usually considerable, often extreme. The organ varies in adults under normal conditions from 4 to 10 oz. in weight. The average may be taken as about 5½ oz. (150 grammes, Birch-Hirschfeld[1]). Its average normal dimensions are 4½ or 5 inches in length, 2½ or 3 inches in width, and about 1¼ inch in thickness. The spleen in leucocythæmia has been found to weigh from 10 oz. (in a child) and 12 oz. (in an adult), twice the normal weight, to 16 or 18 lb., or more than fifty times the normal weight. The greatest weights recorded are 18½ lb. (Langley Browne),[2] 16½ lb. (Sizer),[3] 15½ lb. (Mosler),[4] and 14 lb. 15 oz., after the escape of two litres of blood (Koeberlé). It is rare, however, that the size of the spleen is so great. The following table presents the weight of the organ in 72 cases.

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[1] Deutsche Zeitschrift fur prakt. Med. 1874, p. 40.

[2] Lancet, 1877, vol. ii. p. 310. The spleen was excised. The degree of increase in the white corpuscles is not described in the account of the case, but Mr. Langley Browne has informed me that they appeared more numerous than the red, in the blood which was examined during life.

[3] Gaz. Hebd., 1873, No. 48.

[4] Berl. kl. Wochenschrift, 1864, p. 140.

	No. of cases.		No. of cases.
Under 1 lb.	2	8 to 9 lbs.	1
1 to 2 ,,	5	9 ,, 10 ,,	4
2 ,, 3 ,,	5	10 ,, 11 ,,	2
3 ,, 4 ,,	12	12 ,, 13 ,,	2
4 ,, 5 ,,	9	14 ,, 15 ,,	1
5 ,, 6 ,,	11	15 ,, 16 ,,	1
6 ,, 7 ,,	7	16 ,, 17 ,,	1
7 ,, 8 ,,	8	$18\frac{1}{2}$lb.	1

The average weight of the seventy-two cases is a little under six pounds (5 lb. 14 oz., about 2,500 grammes).

The size of the spleen undergoes a corresponding increase. The organ usually enlarges in all directions, so that the normal proportions are preserved, *e.g.*, a spleen weighing 198 oz. measured 18 × 10 × 6 inches. Occasionally it enlarges more in one direction than another, and thus may become long and narrow, as one which weighed 64 oz. and measured 12 inches long by 4 wide. It varies from 6 to 18 inches in length, from 4 to 10 inches in width, and from 2 to 6 in thickness.

The following table shows the length of the spleen in a series of fifty-six cases. The variations in width and thickness prevent the length being a very close guide to the actual size of the organ, but it is of importance as the most significant dimension which can be determined by physical examination during life:—

Length	6 to 7	inches in	3 cases.	Length	13 to 14	inches in	10 cases.
,,	7 ,, 8	,, ,,	2 ,,	,,	14 ,, 15	,, ,,	6 ,,
,,	8 ,, 9	,, ,,	4 ,,	,,	15 ,, 16	,, ,,	3 ,,
,,	9 ,, 10	,, ,,	7 ,,	,,	16 ,, 17	,, ,,	1 case.
,,	10 ,, 11	,, ,,	7 ,,	,,	17 ,, 18	,, ,,	3 cases.
,,	11 ,, 12	,, ,,	9 ,,	,,	18 ,, 19	,, ,,	1 case.

The average length of the whole series is $11\frac{1}{4}$ inches.

The organ when much enlarged occupies, of course, a large portion of the abdominal cavity. It is sometimes free from adhesions, but more frequently is connected with the diaphragm, abdominal wall, omentum, intestines, and left lobe of the liver. The enlargement is commonly uniform, and the general outline of the organ is preserved, the indentations on the inner border being usually exaggerated, and almost always distinct. The surface is smooth, but often presents the evidences of local peritonitic changes—rarely recent rough lymph, more commonly white or yellowish opaque patches, about a line in thickness, and semicartilaginous in consistence. The colour of the unchanged portions is usually dark bluish purple.

The consistence of the spleen is sometimes normal, rarely diminished, often increased. The cut surface is smooth, even shining, and yields little blood compared with a normal spleen. Virchow pointed out that the larger the spleen and the longer the duration of the case, the firmer is the organ and the less the relative amount of blood in it. In the early stage the organ may be soft and contain much blood. The tint of the cut surface is usually greyish or brownish red, sometimes, in old cases, flesh-coloured or pale brown, or even brownish yellow. Its aspect is, to the naked eye, commonly uniform, sometimes marbled by whitish lines, due to thickened trabeculæ, or less vascular lymphoid tissue. The Malpighian follicles are seldom conspicuous; usually they are not to be distinguished. They are often visible in the early stage of the enlargement, recognisable by their paler tint, but are usually only slightly enlarged, and often not of larger size than that which they may present under normal conditions. Occasionally they are distinctly enlarged, but they are less sharply defined. It is evident that the increase in the size of the organ is due to a growth of tissue corresponding in position to the splenic pulp.

In rare cases, however, the Malpighian follicles do enlarge, become conspicuous, and constitute what are practically small growths scattered through the organ. These cases are for the most part those in which the disease commenced as a primary lymphatic gland affection, or as a simultaneous enlargement of glands and spleen. In the primarily splenic leucocythæmia such scattered follicular growths, differentiated from the splenic pulp, are almost unknown.

The spleen rarely presents softening. In one case a small collection of pus was found in the organ,[1] and in another the upper half of an indurated spleen contained a small focus of softening, while the lower half was converted into a vast pouch filled with a soft material, consisting of *débris* of splenic tissue and colouring matter derived from the blood.[2]

In many cases the spleen presents areas of darker or paler colour than the rest of the organ. They may be dark-red, like extravasated blood,[3] or reddish-yellow, or yellowish-white and caseous. These are situated near the surface, and are more or less wedge-shape in form. Some of them are surrounded by a zone of hyperæmia. They resemble precisely (as Dr. King Chambers first pointed out) the change often seen in cases of heart disease, and now known to be the result of embolic infarction; and their origin, from a similar process of vascular obstruction, has been traced in the leucocythæmic organ by Bourdon. The arteries of the spleen are "terminal," *i.e.*, have no anastomoses. These hæmorrhagic and degenerated areas may sometimes be due to thrombosis in the splenic veins. They have been met with in an enlarged spleen associated with simple anæmia.[4] There may be only one or two such areas, or they may be very numerous. In one case eighteen separate infarctions were observed.[5]

Microscopical examination shows that the process of change consists, in most cases, in a simple increase of tissue elements similar to those which exist in the normal splenic pulp. A scraping of the cut surface presents, under the microscope, a large number of red blood globules, leucocyte-like corpuscles various in size and in the character of their nuclei and contents, spindle cells from the trabeculæ, large cells, oval or round, $\frac{1}{500}$ inch in diameter, containing several nuclei, and sometimes giant cells filled with small corpuscles $\frac{1}{3000}$ to $\frac{1}{1500}$ inch in diameter (Drummond). There is also much pigment, black, red, and yellow.

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[1] Virchow's Archiv, v. 59.

[2] Cuffer, Bull. de la Soc. Anatomique, 1876, p. 195.

[3] Bennett, p. 24.

[4] Müller, Berlin klin. Wochenscrift, 1867.

[5] Bryant, Guy's Hosp. Reports, 1866, p. 444.

On examination of hardened sections a great increase in the trabecular tissue is seen, the finer tracts of which are connected with a network of connective tissue cells closely connected, and passing on the one hand into connective tissue of the trabeculæ, and on the other into a fine retiform tissue of nucleated fibres or films. In the meshes of this tissue lie great numbers of lymphoid cells. The connective network furnishes the numerous spindle cells seen in a scraping. The Malpighian bodies, however, are less distinct. In some cases they have appeared atrophied, in others they are indistinguishable from the rest of the tissue. They have been observed to undergo retrogressive changes, fatty and lardaceous.[1] In the latter case I have found that the degeneration involves equally the periarterial lymphoid tissue of which the Malpighian bodies are but local collections. This periarterial tissue may often be seen to be thickened, and Biesiadecki has asserted that an outgrowth from this into the splenic pulp is the essential process. It is certain that the structure of the splenic pulp becomes far more similar to the lymphatic tissue around the arteries and of the follicles than it is in health. But the fact that when there is a great increase in the tissue of the pulp, the follicular and periarterial tissue may remain distinct, is reason for believing that the pulp growth is not due to the extension into it of this tissue.

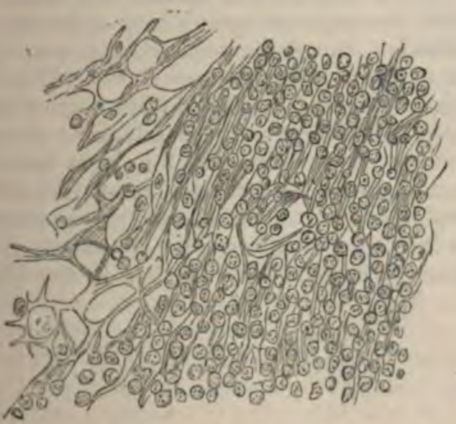

FIG. 4.—Section of spleen in leucocythæmia showing lymphoid cells lying in an irregular stroma, from which, on the left, the cells have been removed by pencilling.

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[1] Böttcher, Thierfelder, and Uhle.

The infarct-like masses present the same histological elements as the spleen. When opaque, the cells within them present fatty degeneration, and are mingled with "compound granular corpuscles," round or oval bodies containing granules and globules of fat. Sometimes only fat cells and the more resistant spindle cells of the splenic trabeculæ are to be distinguished.

In many cases the crystals already described have been found in the spleen. In one case Deiters found them so abundant that on a scraping of the splenic pulp being placed under the microscope, a dozen were visible in every field.

Crystals of cholesterin were found in one case by Goupil. They were arranged in granular masses, visible to the naked eye.[1] On chemical analysis the splenic tissue has been found to contain glutin, glycocoll, hypoxanthin, xanthin, leucin, and tyrosin, but no uric acid.

Supernumerary spleens, "splenicula," always present the same changes as the spleen itself. The spleen has been found ruptured in leucocythæmia, the accident having been apparently spontaneous.[2]

Lymphatic Glands.—In splenic leucocythæmia, the lymphatic glands are enlarged in one-third of the cases. Out of 157 cases, enlargement of the glands, few or many, was found after death or observed during life in fifty-one. (From this number all those cases in which the gland enlargement was an initial symptom have been excluded.) The glands in the abdominal cavity were most frequently affected (in thirty-nine cases), those on the surface next frequently (in thirty-four cases), those in the thoracic cavity least frequently (in eleven cases). The several groups of glands are affected in the following order of frequency:—

Mesenteric	29 cases.	Retro-peritoneal . . .	18 cases.
Cervical	24 ,,	Thoracic	11 ,,
Inguinal	24 ,,	Portal	5 ,,
Axillary	21 ,,	Iliac	3 ,,

The affection of the glands was universal in only five cases. The frequent enlargement of the mesenteric glands is independent of the affection of the intestinal follicles, which may be normal when the mesentery is so changed by the general enlargement of the glands that it stands up from its attachment and is three fingers' breadth in thickness.[3]

The degree of the enlargement of the glands is not often considerable. The individual glands scarcely ever exceed, and rarely reach, the size of a walnut. In this respect a marked contrast is presented to those conditions of lymphatic disease in which the enlargement of the glands is an initial symptom. The glands are usually soft, sometimes very soft, occasionally firm, especially when the enlargement is slight. The surface is smooth and usually free from undue connection djacent structures. On section they are grey or reddish-white, ntly mottled red from vessels. A distinction between cortex edulla is often preserved. Sometimes there are numerous rhages into the substance, especially when the enlargement is the glands soft, and the tendency to hæmorrhage considerable. imes caseation is met with in some of the glands,[1] but it is general or even abundant. Very rarely, some of the glands uppurate. In one case of well marked leucocythæmia with a large spleen, the cervical glands enlarged and suppurated a before death.

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[1] Goupil, Soc. Méd. des Hôp. 1858, Nov.

[2] Seitz, Deutsche Klinik, 1866.

[3] Simon, Thèse, Obs. i.

ler the microscope the glands present an appearance which but little from the normal structure. An adenoid reticulum can en in brushed sections, and in the meshes of this lie great ers of lymphoid corpuscles. There is rarely that increase in the lum which is met with in lymphadenoma. Crystals resembling met with in the blood and the spleen have been found in one n the mesenteric glands (Lauenstein).

mentary Canal.—The gums sometimes present uniform swelling, ccasionally ulceration, which may be gangrenous in character. welling has been found in some cases to be due to a diffused hoid growth in the substance of the gum; in others, the only nised change has been a dense infiltration of the tissue of the with leucocytes.

e lymphatic follicles at the base of the tongue, and the tonsils, he seat of growths of lymphoid tissue occasionally, but far less ently in primary splenic leucocythæmia than in the various of lymphadenoma, and the cases in which they occur are ed by considerable enlargement of the lymphatic glands; as, stance, one recorded by von Recklinghausen.[2] In the same case were soft nodules on the posterior surface of the epiglottis, and, eat numbers, in the mucous membrane of the larynx and trachea; of these were ulcerated.

e mucous membrane of the stomach may be congested, and its y may contain blood, without any breach of surface being le. The collections of lymphoid tissue which lie between the lar glands may be enlarged, and visible as whitish granulations in ubstance of the mucous membrane. Overgrowth of the solitary s may cause them to project conspicuously from the surface of nucous membrane. The affection of these follicles is, however, less frequent than that of the intestinal glands, and never so derable as is occasionally seen in Hodgkin's disease.

cers have been found in the duodenum in two cases:[3] their e is not clear. In neither case was there any affection of the f the intestine. In the other parts of the small intestine, jejunum

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er, Bull. de la Soc. Anatomique 1876, p. 195; Reincke, Virch. Archiv, Bd. 50.
rchow's Archiv, vol. 30, 1864, p. 370.
n Franque, Deutsche Klinik, 1856, p. 462. Mattei, quoted in Gaz. Hebd. 1858,
.

and ileum, the solitary and Peyerian follicles are frequently enlarged, paler than natural, and more prominent. In one case they were described as "thick and fleshy."[1] Rarely, the solitary glands are enlarged, while Peyer's patches are not.[2] Occasionally the lymphoid growth extends beyond the limits of the follicles, and infiltrates, locally, the sub-mucous tissue. Such a growth was found very abundantly in one case by Ollivier and Ranvier; it formed a series of prominences about the size of a pea flattened in the centre, and with the microscope an abundant nucleated retiform tissue, containing round corpuscles, was seen. Similar growths were pigmented in another case, in which they attained a large size, from a pea to a walnut, and existed chiefly in the jejunum.[3] Occasionally one or more masses of growth of still larger size are met with.

These lymphoid growths in the intestine frequently ulcerate. The ulcers have their edges thickened by a similar lymphoid growth, and bear considerable resemblance to tubercular ulcers, for which they were formerly sometimes mistaken. In one case, in which there was a sago spleen, a similar lardaceous degeneration of the openings of the gland ducts in the small intestine was found by Böttcher.[4]

In the large intestine changes are occasionally found similar to those which have been described as occurring in the ileum. The mucous membrane may be softened or congested. There may be sub-mucous extravasations or superficial ulcers. Occasionally, as in the stomach, blood is effused without any recognisable breach of surface. The solitary glands may be enlarged, with or without enlargement of those in the small intestine. They usually become less conspicuous towards the lower part of the bowel. In one case, recorded by Dr. Wickham Legg,[5] there was an immense overgrowth of the solitary glands in the colon, some being as large as filberts. They were surrounded by a zone of redness, and were small in the sigmoid flexure, and inconspicuous in the rectum. These growths are frequently ulcerated, and sometimes the ulceration has the apperance of having been of old standing, and is deep.[6] Occasionally ulcers, apparently unconnected with the lymphatic growth, may be found in the colon.[7] The degree of glandular overgrowth in the intestine presents no regularity when it occurs. The colon may be greatly affected alone, or in conjunction with faucial glands, when the stomach and small intestine are healthy.[8] The vessels in all parts of the intestine may be found filled with white blood corpuscles.

Peritoneum.—In rare cases the surface of the peritoneum has been found to be covered with minute growths, sometimes resembling rcular granulations in appearance and size, and even regarded uch; sometimes larger and evidently lymphoid in character. growths occurred on the peritoneal surface of the intestine in cases; in one case they attained the size of cherries on the , and in one they were pedunculated. Hæmorrhage into the toneal cavity occasionally takes place, and when not fatal by anæmia it causes, the blood usually sets up peritonitis. The se of the hæmorrhage has been various. In one case it was the ture of some vascular adhesions of the spleen; in another it was a cture to let out ascitic fluid. In one case a large extravasation rred in the retro-peritoneal connective tissue.[1]

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[1] Benson, Dubl. Journal, 1872.

[2] Craigie; Desnos, L'Union Méd. 1868; Neumann, Arch. d. Heilk. vol. xi.

[3] Lauenstein, Deut. Arch. f. kl. Med. 1876, Bd. 18.

[4] Virchow's Arch. Bd. 37, 1869 p. 163. [5] St. Barth. Hosp. Rep. vol. xi. p. 68.

[6] Wilks, Guy's Hosp. Rep. 1855, p. 361.

[7] Reineke, Virchow's Archiv, 1870, Bd. 50, p. 339.

[8] Wickham Legg, loc. cit.; Ollivier and Ranvier, Arch. de Physiologie, 1869, p. 416.

Liver.—The liver is frequently diseased in leucocythæmia. Out 109 cases in which the state of the liver was noted, during life after death, it presented some abnormal condition in eighty-seven. in twelve other cases a post-mortem examination was made, and abnormal appearance would probably have been mentioned. A bid state was thus mentioned in 87 out of 121 cases. It seems refore correct to state that the liver is abnormal in at least two-s of the cases of splenic leucocythæmia.

The size of the liver varies. In three cases only was it below the mal, and in these the organ was the seat of cirrhosis, a complication bably accidental. In the remainder, the organ was enlarged. The ee of enlargement varied from a little above the normal to four es the normal. The following table represents the weight of the an in thirty-six cases in which this was noted:—

Between 4 and 5 lb. in	3 cases.	Between 9 and 10 lb. in	1 case.
„ 5 „ 6 „ „	6 „	„ 10 „ 11 „ „	1 „
„ 6 „ 7 „ „	11 „	„ 12 „ 13 „ „	1 „
„ 7 „ 8 „ „	4 „	„ 13 „ 14 „ „	1 „
„ 8 „ 9 „ „	8 „		

The heaviest weight recorded is 13¾ lb.[2] The average of the thirty-cases is a little over 7 lb. (115 oz.; 3260 grammes).

the majority of cases of enlargement (fifty-four out of eighty-n) no morbid appearance was observed. It would probably be rrect to assume that the liver in all these cases was healthy: since know that the most frequent morbid change in leucocythæmia elude ordinary observation, and some of the cases were recorded re the minute changes in the liver were generally known. But many cases of leucocythæmia the liver, even when considerably rged, has appeared to skilful examiners to be normal in struc-e. Virchow has asserted that such enlargement is due to hyper-phy of the liver cells, by which the acini are increased in size. xamples of the size which the liver attained without visible ation in structure may be mentioned—5¾ lb. (Vogel), 6 lb. (Page), (Kœberlé), 6¾ lb. (Da Costa), and 8¼ lb. (Barclay).

The next most frequent change is the existence of minute points or nodules of new growth in the liver. These were found only in thirteen cases, but probably existed in a much larger number, since they can often only be distinguished by the microscopical examination of sections of the hardened organ. With these disseminated growths there is frequently associated a great distension of the capillaries by blood loaded with leucocytes, and this congestion of the liver may be the only recognisable morbid change. It was so in seven cases. The size which the liver may attain may be, in each of these conditions, considerable. In cases of disseminated growths, weights of 6¼ lb. (De Pury), 8 lb. (Patchett, von Franque), 8¼ lb. (Mosler), and 12¼ lb. (Bennett), are recorded; while instances described as congestion have been met with in which the organ weighed 7 lb. (Gordon Jackson), 7¼ lb. (Ollivier), 8½ lb. (Reincke, Bryant), and 13¾ lb. (Walshe).

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[1] Gairdner, Case xii. in Bennett's "Leucocythæmia."

[2] Walshe, Case viii. ibid.

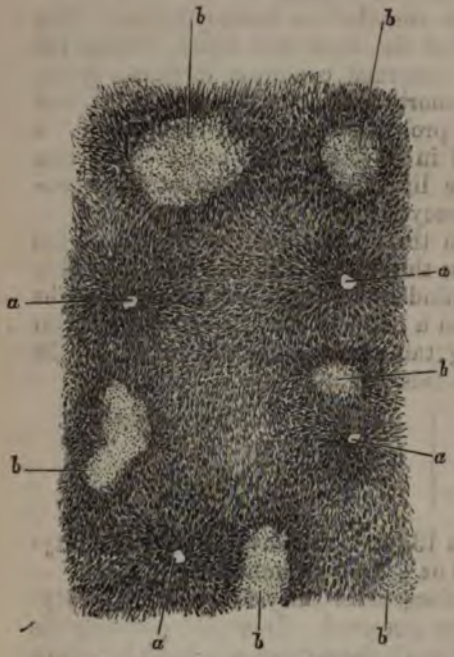

FIG. 5.—New growths in the liver in splenic leucocythæmia, showing their interlobular position. *a a* branches of hepatic vein occupying the centres of the acini. *b b* nodules of new growth between the acini.

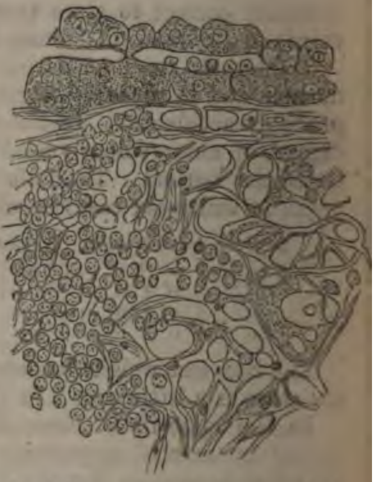

FIG. 6.—New growth in the liver in splenic leucocythæmia. The lymphoid cells have been removed from part of the section by pencilling, so as to show the stroma in which they lie. Above are seen some of the hepatic cells.

The growths in their most common form present the appearance of minute greyish-white granules, the size of ordinary tubercles or larger. The smaller are more or less rounded in form; the larger commonly irregular, according to the conditions of origin and growth.

They may be apparently isolated, and then are commonly situated between the acini of the liver (Fig. 5), or may lie adjacent to vessels, chiefly to branches of the portal vein, and extend along it, sometimes filling up a branch of a portal canal (Fig. 7). They may invade the substance of the acini by extending between the columns of liver cells; less commonly they compress the liver tissue, and may thus become surrounded by a pseudo-capsule of compressed liver-cells. They consist of lymphoid cells lying in the meshes of a nucleated stroma of fibres and films, which can be brought into view by pencilling a section (Fig. 6). The number of leucocyte-like cells they contain, and their position adjacent to vessels, have given rise to the opinion that they arise from emigrated leucocytes, and even that they consist only of extravasated corpuscles. But the presence of a distinct stroma

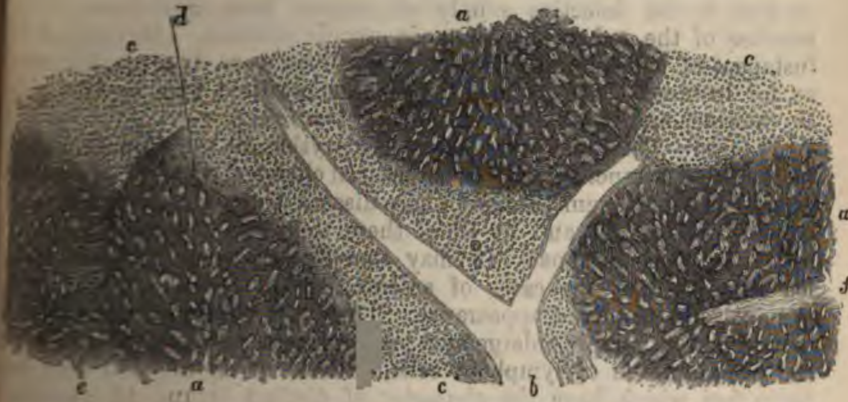

Fig. 7.—Section of the liver in leucocythæmia, showing infiltration of portal canal. *a a* acini of the liver. *b* branch of portal vein lying in the middle of the portal canal. Above its bifurcation is seen the section of a bile duct. *c c* lymphoid growth filling up the portal canal, and at *d* invading the tissue of an acinus. *e* separate centre of growth between two acini. *f* section of branch of hepatic vein in centre of lobule divided longitudinally.

certainly quite different from the coarse laminar stroma in the meshes of which the liver-cells lie, shows that there is something more than extravasation, that there is an actual growth. In support of the view of the local origin of these lymphoid cells is the observation of Cohnheim, in which similar growths were found in the liver in a case of simple enlargement of the spleen without any increase in the white corpuscles of the blood.

The congestion, which is occasionally so marked, consists in the distension of the capillaries with white corpuscles. This may take place in the substance of the acini, so that the rows of liver-cells are separated by masses of white corpuscles contained in capillaries, the walls of which may not be at first visible (Fig. 7). In the interacinal spaces dense accumulations of corpuscles take place, sometimes having the character of a lymphoid growth, pressing on the outer cells of the acinus, causing their atrophy, and sometimes invading the acinus itself, reaching even the centre. These tracts may be visible as whitish ramifications separating the acini. In the substance of the lobule the cells, according to Ranvier, are contained within enormously distended capillaries, but Waldeyer[1] believes that they lie outside the capillary vessels and compress them, as well as the hepatic cells. He believes that even the apparent congestion is really a growth commencing in the connective tissue, in the interstices of which, as von Recklinghausen has shown, the minute lymphatic vessels commence. The lymphoid cells, according to Waldeyer, multiply by division, and enter and distend the minute capillary vessels.

Another frequent change in leucocythæmia is fatty degeneration of the liver-cells. It was conspicuous in ten cases. It is no doubt due in part to the defective supply of oxygen, from deficiency in the number of the red corpuscles, as in simple anæmia. Several of the instances of extreme enlargement of the liver were due to this cause, as, for instance, those in which the organ attained the weight of 9½ lb. (Siering) and 10½ lb. (Bouillaud and Duroziez).

Kidneys.—The kidneys in splenic leucocythæmia may be healthy, or may present parenchymatous or interstitial changes. Out of 111 records of post-mortem examinations in this disease, the state of the kidneys is mentioned in 62, and in 12 of these they are described as normal, in 50 as abnormal. It may therefore be assumed that in at least one-half of the cases of splenic leucocythæmia these organs present some abnormal appearance. The most common changes are, simple pallor; simple enlargement; enlargement with pallor; interstitial accumulation of lymphoid cells, diffused or aggregated into definite foci which have the character of distinct lymphoid growths; fatty degeneration; extravasations of blood in the pelvis; interstitial deposits of uric acid and actual calculi; and lastly, changes indicative of actual "Bright's disease" of various kinds.

In eight cases undue pallor was the only morbid change noted. The exact cause of the pallor has not, in any case, been investigated; but it is probably to be ascribed in part to the blood changes, the deficiency of red and the excess of white corpuscles in the vessels, and in part to undue opacity of the kidney cells. In one of these cases albumen was present in the urine.

In five cases the kidneys were not only pale but large. In one instance the two together weighed 12 ozs., in another 16½ ozs., and in this case they were "mottled." Externally these large pale kidneys are smooth, and the increased size is found, on section, to depend mainly on an increase in the thickness of the cortex. It is probably due partly to distension of capillaries with leucocytic blood, and partly to increased size of the cells of the organ. Albuminuria may be present or absent in these cases. It was absent in the case in which the two kidneys weighed 12 ozs., and is not mentioned in the case in which the two weighed 16 ozs. When albumen has been present in the urine its quantity has been small, and no casts have been found. In three cases the kidneys were large, owing to an increase in the thickness of the cortex, without being pale.

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[1] Virchow's Archiv, Bd. 35, 1866, p. 214.

In many cases the cortex presents white points and lines lying between the tubules. In some, these are most conspicuous in its outer layer. On microscopical examination they are found to depend upon accumulations of lymphoid cells between the tubules. The latter may be separated widely, and their epithelium may undergo degeneration in consequence of the pressure to which they are exposed. According to Ollivier and Ranvier, although these cells apparently lie in the interstitial tissue, they are really contained within greatly dilated capillaries. Virchow and Waldeyer, on the other hand, maintain that they are outside the capillaries in the interstitial tissue, where they are formed by nuclear proliferation, and that they are really lymphatic growths and resemble those of the liver. Virchow pointed out that the accumulations are found within the Malpighian bodies as well as in the interstitial tissue. These cells may undergo fatty degeneration, and minute foci of suppuration may result.[1]

Whether these minute accumulations of cells are growths or not, there occur, in many cases, aggregations of lymphoid cells which have all the characters of lymphoid growths. They may be small, resembling grey granulations in size, or may attain the diameter of a quarter of an inch. Such distinct growths were described in ten cases. They are greyish-white in colour, rounded, and sharply limited from the adjacent kidney substance. They are usually most abundant in, and often confined to the cortex, and sometimes appear to arise in the neighbourhood of the glomeruli. On the surface, the position of the larger is usually marked by a prominence beneath the capsule, sometimes, when there is parenchymatous swelling of the organ, by a depression. The growths, when small, bear, as in the liver, considerable resemblance to miliary tubercle, and were described as such by some of the earlier observers. Under the microscope they consist of lymphoid corpuscles massed together, and on the removal of these a retiform stroma may commonly be found. The kidneys in which these growths occur may be normal in size, but are more commonly enlarged, sometimes to a considerable degree. In one case the two kidneys weighed together 13¼ ozs. The enlargement depends, however, only to a slight extent on the growths. It is due in chief part to the parenchymatous swelling of the organ and capillary distension, which often coexist.

Pronounced fatty degeneration was the conspicuous morbid appearance of the kidney in a few cases. It is probably, when not due to accidental causes, to be ascribed to the anæmia, and is to be regarded as a further stage of the pale enlargement which is so frequent.

In several cases there were accumulations of uric acid between the tubules of the kidney. It will be seen that there is, in this disease, an excess of uric acid present in the blood, and it is not therefore surprising that it should be deposited in the excretory organ. In two cases renal calculi were present. In several cases in which a hæmorrhagic tendency existed, extravasations were found beneath the lining membrane of the pelvis of the kidney.

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[1] Ollivier and Ranvier, Gaz. Méd. de Paris, 1867, p. 365, and Arch. de Physiologie, 1869, p. 416.

Some of the changes above described, if they progressed further, would give rise to a condition which might merit the name of Bright's disease. Actual Bright's disease was present in six cases, but would appear, in most of these, to have been due to extraneous causes, and must therefore be regarded as a complication of the disease, and as such it will be described.

The supra-renal bodies have been found diseased in three cases of leucocythæmia. In two they were enlarged and softened: in one[1] they were the seat of caseous change, the result, it was believed, of the degeneration of extravasated blood. In one case[2] in which the organs were large and mottled, there was some bronzing of the skin.

The thymus gland has been found enlarged in several cases,[3] in one considerably. Its enlargement appeared to be due to a lymphoid growth. The affection is not so considerable or so frequent as in cases of Hodgkin's disease. In one case it was found to contain crystals, such as occur in the spleen and blood. The *thyroid* gland is also occasionally enlarged; it was so in five cases.[4] In one case the left lobe was large and soft, and contained a quantity of semi-purulent fluid.

Heart.—Effusion into the pericardial sac is common. The quantity of fluid is not great, and there are usually no signs of pericarditis. The effusion is part of a universal œdema. In the rare cases in which traces of pericarditis have been found, it has usually been distinctly due to an intercurrent malady, as in one case to variola. The effusion presents the normal characters, being rarely, if ever, hæmorrhagic. Hypoxanthin has been found in it in one case by Salomon. Growths on the visceral layer of the pericardium were found by Wolffhügel, isolated and conglomerate masses, the size of peas, consisting, under the microscope, of roundish granular cells the size of white corpuscles.

In several cases extravasations of blood were found beneath the pericardium on the surface of the heart. By the degeneration of the effused blood they may leave pale slightly prominent patches, and similar spots have been observed on the trabeculæ of the left ventricle, and even in the substance of the wall. The capillaries of the heart have been noticed to be crammed with white corpuscles, accumulations of which may exist between the fibres. In one case the lymphatic vessels on the surface of the heart were unduly conspicuous.[5]

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[1] Vogel, Virchow's Arch., Bd. 3, p. 570.

[2] Barclay, Lancet, Jan. 31, 1863.

[3] Hawtrey Benson (Dublin Journal, 1872); Ehrlich; Waldeyer; Simon (Thèse).

[4] Craigie; Biesiadecki; Lanceraux (Atlas); Page (Brit. Med. Journal, 1851); Feltz (Gaz. Méd. de Strasbourg, 1864).

[5] Ehrlich, Inaug. Diss. p. 13.

The muscular fibres have been found, in several cases, to present granular and even fatty degeneration, as in other forms of intense anæmia. The fibres of the heart have, however, been found healthy, even when the white corpuscles were more numerous than the red, and the capillaries of the heart appeared full of them. The degeneration has been observed to be especially intense in the neighbourhood of old extravasations of blood, beneath the peri- and endocardium.

Pleuræ.—Effusion into the pleural cavities is very common in leucocythæmia, sometimes on one side only, sometimes on both. It may be due to pleurisy, to pleural growths, or may be part of a general œdema. Hypoxanthin was found in the effusion by Andrae. Lymphoid growths occur occasionally on the surface of the pleura, as greyish-white tubercles, consisting of lymphoid cells, which are said to be derived from the connective-tissue elements of the membrane.

Lungs.—Disseminated alterations are occasionally found in the lung in leucocythæmia, although less frequently than in Hodgkin's disease. The pulmonary capillaries may become distended with white blood-corpuscles,[1] and these may even occasionally block up vessels, leading to the infarction of wedge-shaped areas, in which congestion, extravasation, and degeneration may occur. Minute scattered hæmorrhages may also result from the capillary obstruction, and the extravasated blood may fill the air-cells, in which red and white corpuscles are seen mingled with a reticulum of fibrin. In some places this extravasation may undergo caseation and closely simulate a tubercular process.[2]

Actual growths may also occur. These are said to begin by an infiltration of the mucous membrane with lymphoid cells, which cause, first, thickening and opacity of the membrane, and secondly, a villous growth from it which may narrow the calibre of the bronchi, and even entirely obliterate those of small size.[3] The cells may infiltrate the wall of the bronchus and extend outside it into the adjacent lung tissue, the elastic elements of which may be observed among the cells. Between the latter a retiform stroma has also been traced; the structure of the growths closely resembling that of the new growths in the liver and kidneys. In the substance of nodules, which appeared uniformly grey, numerous nests have been found, which looked like lymph follicles, inserted into the tissue. Sometimes several such areas may coalesce into large masses. They bear considerable naked-eye resemblance to tubercles, but are distinguished by their usual freedom from caseation, such as would almost certainly accompany tubercular formations of the same size. No doubt most of the deposits described as tubercle by the earlier observers were really of this nature.

Cavities may even result from this lymphoid infiltration, and the resemblance to "tubercle" may thus be increased. Böttcher has found that the walls of the cavities present the remains of lung tissue, and thus it is evident that a process of lung destruction has taken place, and that the cavities are not merely dilatations of bronchi. There is, however, no evidence of such softening and excavation as that which gives rise to tubercular cavities. The smaller cavities are directly related to bronchi, and therefore it seems probable that they arise by ulceration from the tubes, possibly commencing in small dilatations. In several cases in which phthisical excavations have been described, it is probable that they have been of this nature. But the appearances in the lung, as elsewhere, have in one or two cases been those of true tubercle. Œdema of the lung is sometimes present, apparently as part of general dropsy.

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[1] Isambert and Robin, 1856.
[2] Ollivier and Ranvier, Arch. de Physiologie, 1869.
[3] Böttcher, Virchow's Archiv, 1866.

Brain.—The vessels of the meninges are found post-mortem to be distended with pale blood, or pale clot. Obstruction of the meningeal arteries by aggregations of pale corpuscles was found by Dr. Bastian. Minute lymphoid growths in the meninges have been described in one or two cases. (Kelsch, Murchison.) In the brain-substance hæmorrhage into the perivascular sheaths is common. In cases in which the hæmorrhagic tendency is well marked, cerebral hæmorrhage sometimes occurs. Eight such cases are on record. There are almost always multiple hæmorrhages in these cases, and the separate extravasations may be very numerous. In one case there were seven separate hæmorrhages, one of which, 2½ inches long, had evidently been formed by the coalescence of several smaller extravasations.[1] In another case the brain is said to have been "riddled" with minute hæmorrhages, and in one recorded by Dr. Hawtrey Benson as many as fifty separate extravasations were counted. Although there are usually many foci of extravasation of small size, there may be one large hæmorrhage which may burst into the ventricles.[2] Hæmorrhage into the spinal meninges was found by Blache,[3] without any source being discoverable. The only change which has been found in the spinal cord itself is the presence in it of the blood crystals. (Lauenstein).

Skin.—The skin is occasionally the seat of furuncles and ecthymatous pustules, and in rare cases of growths.[4] In the remarkable case recorded by Biesiadecki there were small tumours projecting half a line above the surface of the skin. Some merely infiltrated the superficial, others the deeper layers of the corium. The larger growths extended into the subcutaneous tissue, but were sharply limited, while the limit of the smaller growths was less distinct. They were composed of round cells, the size of blood corpuscles, with ramifying connective tissue cells; many of the round cells had undergone fatty degeneration. Biesiadecki believes that the furuncles occasionally observed may have their origin in similar growths.

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[1] Reincke, Virchow's Archiv, vol. l.

[2] Troisier and Raymond, Bull. de la Société Anatomique, 1873, p. 693.

[3] Bull. de la Acad. de Méd. 1856.

[4] Lauenstein, Deut. Arch. für kl. Med. 1876; Biesiadecki, Stricker's Med. Jahrbuch, 1876, p. 233.

A remarkable subcutaneous growth in the tissues over the sternum occurred in a case recorded by Dr. Shingleton Smith.[1] The growth, although it ultimately infiltrated the sternum and the anterior mediastinum, and extended up into the neck, appeared to commence in the structures in front of the sternum. It was regarded as a primary affection, but this hypothesis seems scarcely tenable, since, although it was noticed before the abdominal enlargement, it was only the size of a walnut when the spleen reached the iliac bone, and the white and red corpuscles were in equal numbers.

Bones.—The bones are occasionally found diseased in leucocythæmia, the change chiefly affecting the marrow. The association of bone-growth and blood-change was first noted by Ranvier,[2] and its occurrence in splenic leucocythæmia has been carefully studied by Neumann and others. The frequency of the affection cannot at present be determined. It was thought to be invariable by Neumann. In many instances since the change has been known, the bones have been examined carefully, and found to be, in all respects, normal. There are, however, at least fourteen cases on record[3] in which a very marked alteration of the marrow was found. The change resembles in many characters that which has been found occasionally in pernicious anæmia, and in some cases of lymphadenoma. The medulla is changed in consistence and aspect, and is increased in quantity. Instead of its normal red colour, it is pale grey, or reddish grey, or reddish yellow, and often "dirty" in appearance. It is diffluent and slimy in consistence, so that a small portion placed on a glass plate will spread over the surface. Sometimes it is almost purulent in aspect, and a collection of fluid may result, closely resembling an abscess. When pale grey, or greenish yellow, and jelly-like, it has been observed to become redder after a brief exposure to the air. In one or two cases the appearance of the medulla has been somewhat different. In a case, for instance, described by Ponfick, it was red and firm, and the section resembled closely a section of the spleen. In it were numerous areas, sharply limited, some dark red, others pale, and believed to be infarctions, from their characters and their similarity to distinct infarctions which were observed in other organs in the same case.

Externally, the affected bones may present nothing to indicate their change, and may be indeed normal, so far as the osseous tissue is concerned. More frequently there is an atrophy of the cancellous tissue, and the compact substance may also be reduced in thickness. In one case, in which the change had existed for a long time, this had occurred, and the bones were enlarged and very soft. (Mosler.) The outer thin shell of bone may finally be perforated, especially in the ribs, and a growth from the interior may invade the periosteum.

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[1] Brit. Med. Journal, 1874, p. 233.

[2] Journal de l'Anatomie et de Physiologie, March, 1867.

[3] Those recorded by Biesiadecki, Huber, Keussner, Mosler (two cases), Neumann (two cases), Ponfick, Schlepern, Ranvier, Kelsch, Foix and Cornil, and H. Wood. This is exclusive of the cases in which there was not marked leucocythæmia.

Under the microscope, when the change is considerable, the fat cells are seen to have for the most part disappeared, and the tissue appears to consist of the same elements as are seen in the blood—great numbers of lymphoid cells and red blood corpuscles. Besides the cells resembling leucocytes in size and aspect, there are others, smaller, containing a large nucleus, and resembling the cells of lymphatic glands. Sometimes cells are seen larger in size, and containing several lymphoid corpuscles. In some cases intermediate forms between the white and red corpuscles have been observed; but in other cases they were not more abundant than in the blood, and in some they could not be found. Many cells containing red blood corpuscles were found by Ponfick, in a case of leucocythæmia, in marrow which appeared healthy.

The vascular structure of the medulla is sometimes normal in character; more commonly it is changed. The vessels are fewer than normal; the ordinary capillary net-work is absent (Neumann), and the vessels which can be distinguished are chiefly arteries, the walls of which are often infiltrated with leucocytes. They usually contain many white corpuscles, and may be visible to the naked eye as whitish spots and lines. In several cases the minute crystals, which are often so abundant in this disease after death, have been found in the changed medulla. In most instances in which the change was considerable, all the bones examined presented it, although it was usually more marked in some than in others. In those bones which possess much spongy tissue—the ribs, sternum, vertebræ—it was most considerable; but it has also been well marked in the diploë of the cranial bones, and in the long bones—humerus, femur, tibia, and fibula. In a case described by Ponfick the affection of the different bones was not uniform—some were scarcely at all diseased; others, as the tibia, considerably altered.

Retina.—Changes in the retina are frequent in leucocythæmia. The vessels are the parts chiefly affected, the most conspicuous change being the occurrence of numerous extravasations of blood. The larger hæmorrhages are situated in the outer layer of the retina, especially in the layer of rods and cones, and in places may even bulge the membrana limitans externa, and, when large, may effect a detachment of the retina from the choroid. Smaller hæmorrhages are situated in the inner layers, chiefly, in some cases, towards the periphery of the retina, and occur especially in the inner granule layer, and in the ganglion cell layer. In some cases the hæmorrhages have been distributed equally in all layers. Those in the fibre layer have a tendency, when small, to extend between the fibres in the direction of least resistance. The seats of old hæmorrhages are marked by brownish pigment. The capillary vessels are usually filled with white corpuscles, which have accumulated in them, and which in places distend them; in the vessels a little larger than capillaries the red and white corpuscles are present in the proportion usual in the case. Adjacent to the capillaries, red corpuscles lie in the tissue. The larger hæmorrhages are due to rupture of vessels, which were found by Roth to present distinct fatty degeneration, especially in the periphery. The larger vessels may be seen to be surrounded with lymphoid cells. Lymphatic growths were found in the retina, in one case, by Leber; and he was inclined to ascribe the hæmorrhages in all cases to the presence of these growths; but with this, as Reincke pointed out, the character of the hæmorrhages is scarcely compatible. The structural elements of the retina are usually little changed, except in the neighbourhood of the hæmorrhages. Adjacent to these, degeneration of the nerve fibres, and swelling of the fibres of Müller, have been observed. Where the hæmorrhages are very large, these degenerative changes may result in almost complete destruction of the retina at the spot. The choroid is usually little changed, but lymphoid growths have twice been found in it.

SYMPTOMS.—The pathological changes which underlie leucocythæmia have been seen to be profound and widely spread through the organs and tissues of the body. The symptoms which those changes produce, and which characterise the disease, are correspondingly considerable in degree, and extended in range. They may be referred to the four sets of changes which are to be traced in a developed case of leucocythæmia; the change in the blood, the enlargement of the spleen, the enlargement of the lymphatic glands, and the disseminated changes, due to blood stasis and to growth, which occur in various organs. Thus, as a consequence of the blood change there are pallor, dyspnœa, languor, headache, and other nervous symptoms; effusion into the subcutaneous tissue and serous cavities; serous fluxion from the mucous membranes; and hæmorrhage in various situations. The splenic enlargement leads to abdominal distension and to the presence of a palpable tumour, to local pain, and to pressure effects on the functions of the stomach and lungs. The enlargement of the glands leads to various local tumours, and sometimes to symptoms due to their pressure, internal and external. Lastly, the changes in organs give rise to symptoms, often troublesome enough, connected with the mouth, throat, stomach, intestine, liver, kidneys, lungs, and eyes.

It will be convenient to consider first the early symptoms of leucocythæmia, and then the symptoms of the developed disease in greater detail as they are produced by the affection of the several tissues and organs.

Early Symptoms.—The early symptoms of leucocythæmia vary considerably, and their character is influenced largely by the preceding conditions of health. An analysis of 108 cases in which the early symptoms were noted shows that those which occur most frequently are due to the splenic enlargement; abdominal swelling, or a distinct tumour in one half of the cases; splenic pain in one quarter of the cases. Abdominal enlargement, or a sense of fulness, was in the majority of cases the first symptom; splenic pain, although a frequent early symptom, is rarely the first to be complained of. Next in frequency among early symptoms are the effects of the anæmia; weakness, languor, pallor, dyspnœa. Next to abdominal enlargement, weakness was most frequently the first symptom complained of. Digestive disturbances are common. In one or two cases excessive appetite was noted early in the disease, and in a few cases, vomiting or diarrhœa. In a considerable number of cases hæmorrhages occurred soon after the commencement, generally from the nose, rarely from the bowel, stomach, or uterus. In other cases œdema occurred. In a few cases epistaxis or œdema of the feet was the first manifestation of the disease.

The following is the order of frequency of the *early* symptoms, beginning with the most frequent: abdominal distension; an actual tumour; splenic pain; weakness; pallor; digestive disturbance; dyspnœa; hæmorrhage; œdema; cough. Of *first* symptoms the most frequent is also abdominal enlargement, and then follow, in the order of frequency: weakness; splenic pain; an actual tumour; digestive disturbance; pallor.

Blood.—The change in the condition of the blood in leucocythæmia gives rise to some of the most obtrusive and most troublesome symptoms of the disease. Some of these it produces directly, either by the disturbances of the circulation, œdema, and hæmorrhage, to which it gives rise; others, indirectly, by interfering with the functions of the several organs. The alterations in the tint of the blood, and its microscopical characters, have been already described, and the mode of examining it will be further considered in the section on Diagnosis. The pale colour of the blood is conspicuous in a drop obtained from the finger, and is shown strikingly in the paler tint of all the structures which owe their colour to the blood. The skin is white, and sometimes has been compared to marble in appearance. The mucous membranes of the mouth and conjunctivæ are pale, as in simple anæmia. As the anæmia progresses, the defect in the red corpuscles leads to dyspnœa, to disturbance of cerebral function, and to prostration.

Temperature.—In a large number of cases of leucocythæmia there is elevation of the temperature. It is most marked towards the close of the disease, but may exist for a long time previously, and, probably, often commences early in the disease. Occasionally the temperature, previously raised, sinks to the normal just before death.

The frequency with which there is pyrexia is difficult to ascertain, since the existence of slight pyrexia may easily pass unnoticed, and in most recorded cases of leucocythæmia the temperature has evidently not been taken. In only 47 of the cases collected was the state of the temperature recorded. Forty-one were cases of primary splenic leucocythæmia, and of these the temperature was raised in 30, and is stated to have been normal in 11. The remaining six cases were instances of the lymphadeno-splenic form, and of these there was pyrexia in five, and the temperature was normal in only one. Out of the total of 47 cases the temperature was raised in 35 and normal in 12. It seems, therefore, that in three-quarters of the cases of leucocythæmia there is pyrexia. A similar conclusion has been suggested, even more forcibly, by individual observation. Sir William Jenner, who some years ago called attention to the great frequency of pyrexia in the disease, has lately stated that he has found it an almost invariable symptom.[1]

The temperature when raised presents considerable variations, commonly being much higher in the evening than in the morning, rarely highest in the morning. The highest point reached has varied much. In a few cases the pyrexia is said to have been slight, but in only one out of twenty-four pyrexial cases in which the temperature was measured, was the highest point below 100° F. In rather more than half the cases (fifteen) the highest point was between 100° and 102°: in six cases it was 102° or 102·5°, in two others a temperature of 103° was occasionally reached; and in one case the highest point was 105°. The morning fall may be slight, the elevation being continuous, or it may be so considerable as to impress on the fever a remittent or intermittent type. Both these characters are presented in the accompanying tracing (Fig. 8.) Sometimes irregular pyrexial periods alternate with periods in which the temperature is normal. When the variations are marked, the attacks of pyrexia may be accompanied with shivering and sweating, as in hectic and almost as in ague, and this in patients who have never been exposed to malarial influences.

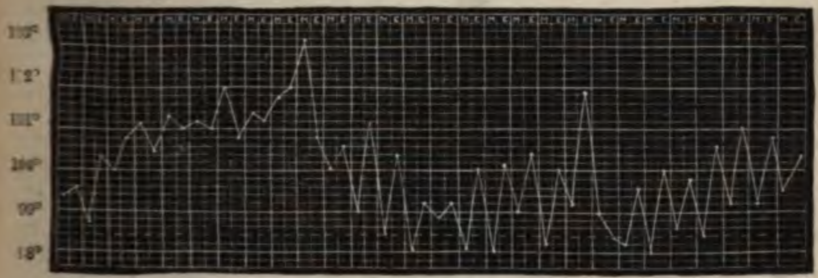

FIG. 8.—Temperature chart of a case of splenic leucocythæmia, showing the two types of pyrexia, the continuous and remittent types. From a case in University College Hospital, under the care of Dr. Wilson Fox.

The cases in which there is most fever are usually those of rapid course, considerable dropsy, and extensive hæmorrhage. The pyrexia does not appear to be related to the size of the spleen, or to the degree of the blood change.

Spleen.—The splenic enlargement which characterises leucocythæmia, as it is the earliest pathological change, also in most cases gives rise to the earliest symptoms of the disease. But enlargement of the spleen does not commonly give rise to symptoms until its degree is considerable; hence, when subjective symptoms arise, the organ is usually large. The enlargement being uniform, its signs are those typical of a splenic tumour. When very slight, increased dulness in the position of the spleen may be its only sign, but when larger, the spleen descends below the ribs, and is borne forward by the costo-colic ligament, on which, in health, its lower extremity rests. It descends in front of this, and of the splenic flexure of the colon, and thus forms a superficial tumour in the left hypochondriac region, having the characters described as those of a splenic tumour in the article on enlargement of the spleen. The notch is almost always distinct. When the enlargement is great, two or three other indentations, which often exist in the anterior margin, may be so exaggerated as to be perceptible to touch. The lower extremity of the enlarged organ may be at the level of the iliac crest, and its anterior edge may reach the middle line. When the increase in size is extreme, the spleen may occupy the whole left half of the abdomen, displacing the intestine, reaching the spine behind, and extending an inch or two beyond the middle line in front. It may descend into the iliac fossa, and even into the pelvis, where its extremity may be detected, behind the uterus, on a vaginal examination (Spencer Wells). The splenic tumour usually moves with respiration, even when large, but occasionally its movement is interfered with by adhesions. When not fixed, it may be altered in position by posture. The degree of enlargement may vary from time to time, partly from variations in the morbid state of the system, and also in dependence on physiological conditions. It has been observed to become larger after a meal,[1] and also to be larger in the morning than in the evening.

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[1] Clin. Soc. Discussion, Lancet, 1876, vol. ii. p. 787.

Friction may sometimes be heard over the tumour, and a friction fremitus felt. A murmur, systolic in time, and analogous to the uterine souffle, may also be occasionally heard in the enlarged spleen. This is not special to leucocythæmia.[2] The enlarged spleen commonly causes an unpleasant feeling of distension, often amounting to considerable distress. This is always greater when the stomach is full, and sometimes exists only after meals. The tumour may be tender on pressure, or may be the seat of considerable spontaneous pain, either dull and constant, from the tension and weight, or sharp and lancinating, in consequence of either temporary acute enlargement, or local attacks of peritonitis. Such stabbing pain is often felt over one part of the spleen only.

The pressure which the spleen exercises interferes with the functions of adjacent organs, especially with that of the stomach. Vomiting and indigestion from this cause are frequent. Interference with the descent of the diaphragm may produce dyspnœa, or increase that which existed before, and the interference with the action of the heart, intensified by the gastric disturbance, may give rise to troublesome palpitation.

Glands.—The enlargement of the lymphatic glands rarely gives rise to symptoms except those of the local swellings. They do not commonly attain a sufficient size to interfere with movement, or to produce symptoms by compressing adjacent parts. Hence the enlargement of the thoracic and abdominal glands commonly escapes notice during life. In rare cases the enlarged mesenteric glands can be felt. The superficial glands give rise to small soft tumours freely movable. In these respects the splenic forms present a marked contrast to the cases of lymphadenosis. I have not been able to find a case of primary splenic leucocythæmia on record, in which even dyspnœa was occasioned by the pressure of the enlarged glands on the air-passages, although in lymphadenoma this is a not uncommon cause of death. During the last few days of life in splenic leucocythæmia, as in Hodgkin's disease, the glands often present a remarkable diminution in size.

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[1] Dr. George Johnson, Lancet, Jan. 1870.

[2] It was long ago pointed out by Dr. Hare that such a murmur is distinctive of a splenic from a renal tumour.

In the cases which have been spoken of as the lymphadeno-splenic form, the enlargement of the glands occurs early and is considerable. It may give rise to the symptoms and effects seen in Hodgkin's disease and described in the next article.

Bones.—The alteration in the marrow of bones which occasionally occurs in leucocythæmia is not usually manifested during life by any symptoms. In rare instances, when the degree of change is considerable, the bones have been tender and even painful. In a case, for instance, recorded by Mosler, there was great tenderness over the bone chiefly affected, but no symptoms pointing to other bones in which the affection of the medulla was slighter. The tenderness may sometimes be extreme. When the medullary change is advanced, the bone may be swollen. In another case, also recorded by Mosler, pains existed for several years in the sternum, which became ultimately swollen and soft, and there was enlargement of the iliac bones, some of the ribs, and the left trochanter.

Circulatory System.—The heart may be displaced upwards by the abdominal enlargement and may even come to be almost horizontal inposition. The displacement may be upwards and to the right, as in a case recorded by Dr. Walshe, in which the heart's apex was within an inch of the sternum and three inches of the clavicle. The heart suffers in its function as in simple anæmia. It is frequently slightly dilated and its action is disturbed by the anæmia and pressure, so that palpitation is a very common symptom. The pulse is, in most cases, unduly frequent; and the frequency, during the attacks of functional disturbance, may be extreme, 160 or 170 per minute. A venous hum is often to be heard in the veins of the neck, and a systolic murmur at the base of the heart. A systolic apex murmur has also been frequently noticed, and regarded as mitral, but probably in some cases was merely ventricular in origin.

The peripheral circulation may be disturbed, partly by the blood state, partly by the cardiac weakness, partly by the accidental obstruction of capillaries by aggregations of corpuscles, partly by changes in the vascular walls.

Effusion of serum into the cellular tissue and cavities of the body is very common; indeed it is almost invariable in the later stages of the disease. It may occur, even in an extreme form, from the blood state, but its amount is often increased by pressure on the veins by enlarged glands. The subcutaneous œdema may be so great as to separate the epidermis, or rend the cutis as in lineæ albicantes, and into the spaces thus formed blood may be effused. The œdema may extend to the scrotum and trunk. Ascites is frequent, and sometimes is so great as to necessitate paracentesis. When the effusion is considerable, there are usually enlarged glands pressing on the portal vein. Hydrothorax may occur, with or without inflammation of the pleura. When alone it is usually slight; when there is pleurisy the amount of effusion may be large, and the patient's life—since the effusion is often double—may be seriously imperilled by it. Hydropericardium may also occur, but rarely in sufficient degree to attract attention during life.

Hæmorrhages, external or internal, are very frequent in leucocythæmia. Their precise mechanism is unknown. It has been customary to refer them to a degenerated state of the blood vessels, the consequence of the anæmia, and to this must probably be added the occurrence of local obstructions to the circulation, due to the blockade of vessels by aggregations of white corpuscles, the watery state of the blood, by which its flow is facilitated, and the imperfect character of the coagula. The hæmorrhages may occur in a similar degree in cases of simple splenic tumour without any excess of leucocytes in the blood. Whatever be their mechanism, the frequency of the hæmorrhages constitutes them a very salient feature of the disease. They may occur so frequently, and so abundantly, that the case may be regarded as one of the hæmorrhagic diathesis. They may be among the earliest symptoms. Epistaxis has been noted in one case for six years before death.[1] In other cases the hæmorrhage only occurred towards the last. By the additional anæmia thus caused, the downward progress of the patient is often accelerated.

The frequency with which hæmorrhage occurs has been variously stated; most authorities consider that it is present in three-fourths of the cases. In those I have collected it is mentioned less frequently. Out of 150 cases of which the record was sufficiently full to lead to the expectation that, if present, the symptom would have been mentioned, hæmorrhage, external or internal, occurred in eighty. The most frequent source of hæmorrhage is the nose. Epistaxis is often profuse and difficult to control, and may be the immediate cause of death. It occurs frequently, and in some cases has occurred daily. The next most frequent source of hæmorrhage is the bowel, commonly in association with diarrhœa. It may result from ulceration, or may take place from the surface of the mucous membrane: no breach of surface being visible post-mortem. Hæmatemesis is less frequent, but was present in several cases, and in one or two was the case of death. As already stated, it may also occur without ulceration. The phagedenic ulceration of the gums is sometimes accompanied by hæmorrhage. Hæmoptysis and hæmaturia are less common occurrences, but have been noted in several cases. Hæmorrhage from the uterus occasionally occurs in women. Traumatic causes may also, as in hæmophilia, give rise to hæmorrhage very difficult to arrest. Most cases of excision of the spleen have proved fatal from this cause, and in two cases extraction of a tooth gave rise to extreme hæmorrhage, which in one was fatal.

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[1] Patchett, Lancet, May 18, 1872.

Internal hæmorrhages also occur. Of these the most frequent, although the least important, are the cutaneous extravasations. In some cases, however, they are serious from their size and number. They may also occur beneath the conjunctiva. Next in frequency, and first in importance, are extravasations into the cerebral substance. These occurred and caused death, with symptoms of apoplexy, in eight cases. Extravasations into the muscular substance of the heart were noted in one case, and in one extensive extravasation took place into some of the larger joints.[1] Hæmorrhage may occur into the connective tissue beneath the skin, or between the muscles. In one case an extensive extravasation occurred among the muscles of the back, forming a large tumour, which was opened, and the patient died in consequence.[2] In another case a large extravasation occurred behind the peritoneum. The peritoneal cavity was the seat of hæmorrhage in several cases, in one from some vascular adhesions, which became torn by the weight of the spleen: in one from thickened omentum; in one from a puncture in paracentesis; in several from excision of the spleen.

Respiratory System.—In nearly all cases of leucocythæmia there is shortness of breath, and the dyspnœa is mainly to be attributed, as in simple anæmia, to the diminished capacity of the blood for oxygen. There is always a diminution in the number of red blood corpuscles, which are the chief means for the conveyance of the oxygen from the lungs to the tissues. It is also in part to be ascribed, as Trousseau pointed out, to the pressure exercised by the enlarged spleen on the thorax; and the dyspnœa may thus be intensified by the recumbent posture, and by a heavy meal. It may also be in part due to changes within the lung tissue,—minute growths or obstruction of the lung capillaries by white corpuscles. The dyspnœa is commonly not noticed during rest, but becomes intense on exertion, and in extreme cases the slightest movement produces an agony of breathlessness.[3] Pettenkofer and Voit[4] found that during rest neither the oxygen absorbed, nor the carbonic acid given off, differed from that absorbed and given off by a healthy man. Hence, as Mosler points out, the blood corpuscles probably, at rest, perform an extra amount of work, analogous to that which, in health, they perform during muscular exercise, but there is no reserve of capacity for carrying oxygen which can be used on the occasion of muscular exercise, and hence the dyspnœa on exertion. It is possible that the white corpuscles may to some slight extent assist the red as oxygen carriers. In one case of leucocythæmia the diminution in the number of the red was greater than (according to other observations) is compatible with life, but there was such an increase in the white corpuscles that they were more numerous than the red. Lymph has been observed to redden slightly on exposure to air.

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[1] Klemme, Inaug. Diss. Cassel, 1863; Schmidt's Jahrb. vol. cxxxi. 1866, p. 173.

[2] Vidal, Gaz. Hebd. 1856, Obs. II.

[3] As in a case recorded by Dr. Day, Clin. Hist. and Comment. p. 184, in which towards the last "scarcely any red corpuscles could be seen in the blood."

[4] Zeitschrift f. Biologie, 1869.

Other causes sometimes increase the amount of dyspnœa. Bronchial catarrh is common. Various lung and heart complications may co-exist; pleurisy, hydropericardium, degeneration of the heart's substance. In cases of the lymphadeno-splenic form, glandular tumours in the posterior or anterior mediastinum may compress the lungs or bronchi.

Cough is frequent, due to the various sources of irritation of the pneumogastric, and it may be an early symptom of the disease. The voice may become weak and hoarse.

Alimentary Canal.—The mouth and throat are occasionally diseased. In cases in which the hæmorrhagic tendency is well-marked, the gums may bleed without breach of surface. Lymphoid growths in the gums and pharynx may lead to ulceration, and a low form of stomatitis and pharyngitis, which has been described by Mosler as *stomatitis* and *pharyngitis leukæmica.* But such growths are very rare in splenic leucocythæmia. Their relation to an excess of white corpuscles in the blood is very doubtful. Occasionally a phagedenic ulceration of the gums has been present, analogous to that of scurvy, and Mosler suggests that these low forms of stomatitis may be in part due to the contamination of the blood by the abnormal substances which have been found in it on chemical examination, and which may be deleterious.

Gastric Symptoms.—The appetite is usually little affected until the later stages of the disease, when it is often much diminished, and the inability to take food contributes to the increase of the cachectic state. In a few cases, at the outset of the disease there has been excessive appetite. Thirst is occasionally a prominent symptom, especially when there is pyrexia. The direct effect of the spleen on the stomach may interfere with the gastric functions. Pressure on the viscus often leads to vomiting of food, or, when food is retained, to its imperfect digestion. The pressure, it is also to be noted, reaches its maximum after a meal, when its effect is most injurious. The spleen has been observed at that time to increase in size. The stomach derives part of its blood supply from the splenic artery, and the variations in the blood supply to the spleen will be attended by variations in the blood supply to the stomach. Vomiting, however, although occasional, is not often a troublesome symptom. It was so in only five out of 160 cases. Hæmatemesis is an occasional symptom, and may be the immediate cause of death. It occurred in eight cases.

It may occur without any visible breach of surface, probably from rupture of minute vessels (when the blood state favours hæmorrhage) during the sudden congestions to which the stomach is liable in this disease. It sometimes, however, is the result of the ulceration of lymphoid growths.[1]

Intestines.—The bowels are frequently disordered. Rarely there is constipation, and chiefly in the early stages. Diarrhœa is extremely common. It is occasionally an early symptom, but more frequently occurs during the later stages, and is a very common cause of death. It is usually simple, but occasionally dysenteric, with much mucus and tenesmus, and sometimes may be accompanied by considerable hæmorrhage. When simple, it may be due to intestinal catarrh, or may be the accompaniment of lymphatic overgrowth in the follicles, with or without ulceration. The occurrence of these lymphoid growths, however, by no means involves diarrhœa. They may be accompanied by no intestinal symptoms, or there may be constipation with tenderness of the bowels. On the other hand, just as in the stomach, considerable hæmorrhage may occur without breach of surface being visible after death.

The Liver, as already stated, is frequently enlarged in leucocythæmia, and the enlargement may be great. Its nature cannot be recognised during life, since the new growths, when they occur, are of so small a size as not to be recognisable on external examination. The enlargement may add considerably to the patient's discomfort, increasing the distension of the abdomen, which results from the splenic tumour. It is not often that there is any recognisable interference with the liver functions. Jaundice has been present in a few cases, but when considerable has appeared to be due to the compression of the bile duct by enlarged glands in the hilus of the liver. A slighter degree of jaundice has been present in several cases—a peculiar yellowish tint, to which the term "icterus lienalis" has been applied. Mosler suggests that it is probably due to resorption in the liver, just as in cirrhosis, on the ground that interstitial changes are so frequent in the liver in leucocythæmia. But the tint, in distinct degree, may be observed in cases of splenic anæmia, where there is no excess of white corpuscles in the blood, or any enlargement of the liver, or evidence of disseminated growths in it, or other organ. The enlarged spleen cannot itself cause jaundice, and its influence can only be exerted by means of the blood. There is always, in these cases, profound anæmia. Hermann has shown that there is such a thing as an anæmic icterus; for after water had been injected into a vein he found bile in the blood. In such a case the change in the blood probably lessens its power of destroying or changing the bile pigment absorbed from the intestine, which the blood is known in health to possess. In a case of considerable jaundice, observed by Mosler, bile acids were found in the urine, but their presence in the blood appeared to have no prejudicial influence, such as might be inferred from their power, which Dr. G. Harley observed, of destroying red corpuscles. The degree of leucocythæmia lessened instead of increasing during the existence of the jaundice.

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[1] Mosler, Virchow's Archiv, vol. lvi. 1872, p. 25, and vol. lvii. p. 532.

Ascites is a common symptom in leucocythæmia. Its occurrence is always furthered by the tendency to serous effusion, and sometimes a slight ascites has appeared to be only part of the general dropsy. In other cases it has been evidently increased by some cause of portal congestion. Enlarged glands in the portal fissure may press on the portal vein. In other cases the obstruction may, as Ollivier and Ranvier suggest, be due to the accumulation of white blood corpuscles in the capillaries of the liver, an accumulation which has been repeatedly demonstrated after death. It is also possible that peri-vascular accumulations of corpuscles may compress many of the portal venules, and interfere with the flow through them. Ollivier and Ranvier suggest that the accumulation of corpuscles in the peritoneal radicles of the portal vein may prevent the effusion of serum in some cases, but this is an assumption which is destitute of proof or of any probability from analogy. The degree of ascites is usually moderate, but may be so great as to necessitate paracentesis. Hæmorrhage may occur into the cavity of the peritoneum from many sources, and the extravasation of blood may give rise to considerable, even fatal, peritonitis. Local peritoneal inflammation may also occur from the splenic enlargement. General peritonitis, from any other cause than hæmorrhage, is very rare.

Urine.—The quantity of urine varies in different cases, and at different periods of the same case. It is occasionally diminished from the first. More frequently it is increased towards the end. It is commonly very acid in reaction, and has a medium or high specific gravity, 1020—30. Deposits of lithates are frequent, especially in febrile cases. Under the microscope, crystals of uric acid and of oxalate of lime may often be seen.

The amount of chlorides was found by Schmuziger to be usually from 100 to 150 grains. In Vogel's case the quantity was larger. The phosphates and sulphates were found in Vogel's case to be of normal quantity. The amount of urea excreted probably differs little from that of health, varying with diet, rather than in consequence of the morbid state. Jakubasch found it diminished in one of Mosler's patients, and Salkowski found it on the average less than in health: Thierfelde and Uhle, on the other hand, found it much increased. So, also, did Berrell.[1] In Schmuziger's case, the daily excretion was a little below the normal, varying from 200 to 400 grains.

The amount of uric acid excreted is always increased, often considerably. Ranke[2] found it increased by one-half, from 0·648 to 0·915 parts per thousand. Pettenkofer and Voit found the average of five normal men to be 0·872, while that of a leucocythæmic patient was 1·424, an increase of 64 per cent. Ossikovsky also found an increase, the excretion being on an average about 22 grains, (1·5 grammes) in the 24 hours. A similar increase was noted by Schmuziger and by Berrell. In the case of the latter, a boy of 17 excreted 18·28 grains daily, 1·50 parts per thousand—the average for a boy of the same size and weight, on scanty diet, being, according to Parkes, only six grains. Bartels found an enormous increase in one case, the daily excretion being 4·2 grm., about 60 grains, and mentions another case of leucocythæmia with an enormous spleen, in which concretions of uric acid were frequent, and death occurred after renal colic and anuria of three days duration.[1] Virchow and Ranke ascribed the increase in the uric acid to the functional over-action of the enlarged spleen, since uric acid has been found in many cases in the splenic pulp. Mosler, however, did not find that the increase of the uric acid was constant, but thinks that it occurs chiefly during the febrile paroxysms, and refers it simply to incomplete oxidation during the pyrexia. Salkowski found the increase, although in one case considerable (2·0 grm.), was in another slight. He showed that the increase, even when permanent, is not proportioned to the size of the splenic tumour. This has also been clearly established by Mosler in a case of splenic anæmia, in which there was a distinct increase. Reincke thought that the urea and uric acid are in normal relation to one another, but that the uric acid is increased. He found the urea 2·5 to 2·8 per cent., and the uric acid 0·06 to 0·11 per cent. Hoffmann found the proportion to be 1 to 29 or 36, the uric acid being 0·08 to 0·1 per cent., and the urea 3·07 and 3·22 per cent. In Berrell's case the proportion was 1 to 27.

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[1] Med. Times and Gazette, I. 1868, p. 285.

[2] Virchow's Archiv, Bd. 5, p. 108.

Berrell found the chlorides to be less, and the sulphates and phosphates more abundant than the average.

Hypoxanthin has been found in the urine by Körner, Salkowski, and Andrae, but only in small quantities. It seems not to be invariably present. Cases in which it could not be discovered have been published by Salkowski, Huber, Reincke, and Salomon. The last observer found a body allied to hypoxanthin, and Andrae thought that he found xanthin.

Lactic acid was found in one case by Körner, but could not be discovered by either Salkowski or Salomon. Salkowski found very small quantities of formic acid.

Albumen is present in the urine not unfrequently, apart from the complication of concurrent Bright's disease. In several cases in which it was present in the urine, the kidneys post-mortem were found to present the morbid appearances described in the section on the Pathological Anatomy: increased size, with or without undue pallor; interstitial lymphoid infiltration; and disseminated growths. In none of these cases, however, was the albumen abundant, and in no case were casts met with. In many cases these structural changes existed without any alteration in the urine. In one case in which a trace of albumen was met with, the kidneys were found to be normal. In five other cases in which there was no post mortem examination, albumen was present in the urine without casts or other signs of Bright's disease. In one it amounted to a sixth, and in one or two a few blood discs were found. In rare cases blood is present in the urine in considerable quantities.

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[1] Deutsche Klinik, 1856, p. 23.

Organs of Generation.—Menstruation is usually irregular and commonly arrested as the disease progresses; often early in its course. Occasionally the catamenia are at first excessive, amounting even to profuse menorrhagia. When abundant, the discharge may be dark and fœtid. When there is amenorrhœa, the monthly period may be marked by some other symptoms, as by an increase in the splenic pain or by epistaxis.

Sexual power in men is apparently not specially affected. In two, cases[1] a peculiar erection of the penis has been noted, coming on suddenly, unattended by any sexual feeling, lasting a week or two, and then subsiding. It has been supposed to be due to thrombosis in the corpora cavernosa.

Nervous System.—The cerebral functions suffer in a very striking manner from the altered blood. Langour and physical depression have been noticed as early symptoms in some cases, and are almost constant in the later stages of the disease. Mental failure and loss of memory are occasional symptoms. Noises in the head or ears, commonly intermittent and of arterial origin, are very common. Headache is frequent and often severe. Vertigo is occasionally complained of, and may be a very marked symptom. It may be extreme on any sudden movement, and in one case it was associated with attacks of sudden heat and sickness. When intense, it may be connected, as it so often is, with aural disturbance, of which there is sometimes independent evidence. Delirium has been present in a few cases, and has been ascribed to plugging of the minute vessels of the cerebral cortex with white blood corpuscles; a condition found in one such case by Dr. Bastian. A more chronic form of mental derangement has been noted in two cases; in one of which (recorded by Charcot and Robin) suicide was committed. Coma may occur, it is said, from the effects of the anæmia alone. The graver cerebral symptoms are probably due to capillary obstruction, or to the small hæmorrhages, in part the result of such obstruction. More definite symptoms of local disease, hemiplegia and profound coma, are usually due to extravasation of blood.

Organs of Special Sense. Ear.—The very common noises in the ear have been already alluded to. Deafness is also common, especially towards the end. It sometimes comes on suddenly, and has been thought to be due to intra-aural hæmorrhage. The fact has not yet been demonstrated by post-mortem examination.

Eye. Leukœmic retinitis.—The occurrence of a special change in the retina, visible with the ophthalmoscope, was pointed out first by Liebreich, and the observation has since been abundantly confirmed.

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[1] Longuet, Prog. Méd. 1874, 32; Klemme, Inaug. Dissert., Cassel, 1863; Schmidt's Jahrbuch, vol. cxxxi. 1863, p. 173.

The anatomical changes which occur have been already described. When examined with the ophthalmoscope, the tint of the choroid is seen to be paler than normal, a bright orange. The retinal arteries are narrow, and have the same tint. It may be distinct even by gas-light, and is very striking when viewed with the daylight from a bright cloud, or admitted into a darkened room through a small hole. The most striking difference is in the veins, which are a pinkish or bluish-red, instead of the dark purple colour which they usually present. They may be narrow, scarcely larger than the arteries, or they may be distended, especially when other retinal changes co-exist. The vessels may be accompanied by bands on their sides, white or yellowish, more conspicuous on the veins than on the arteries. The optic disc itself may be normal or may be unduly pale. Occasionally it is slightly swollen and opaque; the vessels upon it being concealed as with a cloud, the margin being gradually lost in the adjacent retina. The retinal tissue may be slightly opaque, but this opacity is secondary to the other changes. Hæmorrhages constitute one of the most striking appearances. They are usually small and most abundant towards the periphery, but they are occasionally of considerable size. They are often striated, the striæ following the direction of the nerve fibres. They may be observed to become paler after a little time, and a succession of fresh ones may be observed.

The centre of many of these hæmorrhages is a white or yellowish-white spot, and sometimes the pale central area may be so large that the aspect is that of a whitish spot with a reddish halo. Similar spots may be observed without any accompanying extravasation. They may be very numerous, and may be grouped around the macula lutea, very much as the spots are grouped in the retinitis of Bright's disease. They are often most abundant towards the periphery. Sometimes they are of considerable size, and then may be traced to the choroid, as in one case in which Becker observed a large white prominence exactly in the position of the macula lutea, and on its margin a zone of red extravasation in which were many white dots. It was evidently a small growth. Subsequently, a second similar spot appeared between the disc and the macula lutea. In one case an extensive hæmorrhage occurred into the vitreous. Hæmorrhages are not always, however, the most conspicuous feature. In one case, which came under my own observation, the retina was the seat of parenchymatous swelling and the veins were greatly distended, and tortuous both in lateral and anteroposterior curves.

The extent to which these changes interfere with sight varies. The disseminated hæmorrhage or white spots will produce very little effect, and the swelling of the optic disc is never sufficient to compress the nerve fibres. When, however, the changes have their seat in or near the macula lutea central vision may be impaired or lost. In Becker's case, for instance, there was a large central defect on the field of vision. The occurrence of an extensive hæmorrhage may cause complete blindness. In one case, red vision by candle-light was complained of.[1]

The Skin is usually pale, the pallor being, as has been said, sometimes a very striking symptom. The tint may be simply white; sometimes a greenish-yellow colour has been observed, the "icterus lienalis" before described. In several, a slight dark pigmentation has been noted, and attention has been called to this by Dr. Greenhow. It may be so considerable as to suggest a resemblance to Addison's disease. The skin is sometimes dry; more frequently there are excessive perspirations, especially when there is hectic fever. Furuncular skin diseases are not uncommon. Petechiæ are frequent, and subcutaneous extravasations may cause soft bluish swellings.[2]

COMPLICATIONS.—Leucocythæmia is frequently associated with other morbid conditions, which have so little connection with the process of the original disease, that they may be regarded as its complications. Some of these, as pneumonia, are so disconnected with the lymphatic disease, or state of the blood, that they may be regarded as accidental. Others, such as fatty degeneration of the liver, or pleural effusion, are extreme degrees of changes which, when slighter, are to be regarded as parts of the lymphatic affection, or its consequences. The separation of some of these from the symptoms, as complications, is to a large extent arbitrary. This is especially the case with the various forms of serous effusion which are so frequent, the anasarca and the peritoneal and pleuritic effusions.

Pleural effusion is a condition very frequently met with. In many cases it is simply part of the general dropsy. In others lymph is formed, and all the signs of pleurisy are present. The effusion is frequently double, and usually occurs towards the last, although it is not often the immediate cause of death. Œdema of the lungs is also common. Hypostatic congestion may occur towards the end. Acute lobar pneumonia is an occasional complication, usually single, rarely extensive, but very frequently fatal. Phthisis is rare. In a few cases tubercles have been found in the lungs, and in a few an ulcerative process in new formations, disseminated through the lungs, has given rise to small scattered cavities, and to the physical signs of phthisis. In most cases, however, this has arisen in lymphatic formations, and the cavities have been, as a rule, small, and the physical signs slight. Bronchial catarrh is common, but rarely serious in its degree.

Pericardial effusion is not uncommon, but actual pericarditis is rare, and the effusion is never great. Weakness of the cardiac walls, due to changes consecutive to the anæmia, degeneration of the fibres and extravasation of blood, may permit dilatation of the heart, and this has been a conspicuous feature in several cases. Valvular disease, when present, is usually due to degenerative changes. In one case aortic, and in several mitral regurgitation, was present.

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[1] Church, British Med. Journ. Sept. 19, 1868.

[2] Simon, Thèse, p. 15.

Peri- and endo-carditis was found post-mortem in one case, but death occurred from an acute specific disease, varioloid, and to it they were probably due.

The disturbances of the circulation which occur in leucocythæmia are to be regarded as symptoms, rather than as complications of the disease. One or two of them have, however, such independent effects, as to merit special consideration.

Thrombosis has occurred in a few cases, but is much less frequent than in Hodgkin's disease, in which the circulation is often impeded by pressure on the venous trunks.

Gangrene of the fingers, the result apparently of defective supply of blood, was noted in one case, but probably preceded the leucocythæmia, and was an accidental complication.[1]

Cerebral hæmorrhage is an occasional and very grave complication. It occurred in eight cases, and in each was the cause of death. The gravity arises from the fact that it depends on the local effect of a general cause, and the condition produced is widely spread through the brain, and the hæmorrhage is therefore generally extensive and often multiple. Its occurrence has no relation to age. The ages of six of the patients were respectively seventeen, nineteen, twenty-two, thirty-five, thirty-five, forty. In several of the cases the hæmorrhagic tendency has been well marked, in others, however, there has been no external hæmorrhage. Its onset has usually been marked by sudden coma, rapidly deepening to death. In consequence of the number of the extravasations, the symptoms have in many cases been from the first general—those of a limited cerebral lesion having been absent.

Cirrhosis of the liver has been met with in a few cases. It has been regarded as an independent and accidental complication, but it must at the same time be remembered that the leucocytal infiltration and growth in the portal canals bears some resemblance, in its minute structure, to that which, in some cases, initiates cirrhosis, and that the adenoid growth elsewhere, may occasionally present a tendency to induration. In one case the kidneys also were large and fibrous.

Fatty livers are often met with. Jaundice is occasionally present, and has been already spoken of. When considerable it is usually due to the pressure of enlarged glands on the bile duct, or is a purely accidental complication. Extreme ascites may also result from the enlargement of the portal glands.

Bright's disease is an occasional complication of leucocythæmia. The most common form is a sub-acute parenchymatous degeneration of gradual onset, the kidneys being large, sometimes pale, sometimes mottled red, and the urine highly albuminous and containing casts, granular and epithelial. A considerable increase in the anasarca often attends it. In one case the cortex presented some atrophy, and in one or two the kidneys were large, white, and presented extreme fatty degeneration. The common form is probably only a further stage of the slight parenchymatous swelling which is so common in the disease, and gives to the organs their large size and pallor so frequently described.

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[1] Simon, Thèse, Obs. 11.

Renal calculi were found after death in three cases, but had given rise to no symptom during life. Their formation is no doubt dependent on the large excretion of uric acid, deposits of which are sometimes observed in the kidney substance.

The morbid states of the alimentary canal are rarely of such independent character as to deserve separate consideration.

The skin sometimes presents pathological conditions, commonly related to the cachexia, or œdema. Furuncles occasionally occur. Pemphigus has been present in one or two cases towards the close. Erysipelas also occurs in the later stages, especially in œdematous parts.

Symptoms of Addison's disease have been noted in a case recorded by Dr. Barclay, in which the bronzing of the skin was associated with enlargement and mottling of the supra-renal bodies.

DURATION.—The insidious commencement of leucocythæmia renders the determination of its duration a matter of much difficulty. A period of ill-health, in many cases, precedes the definite symptoms of the disease. In other cases, the first indications of ill-health only attract attention when the splenic tumour and blood change have made considerable progress. In each instance it may be difficult or impossible to fix the commencement of the disease. In the latter case it will probably be post-dated, in the former, ante-dated. But since the sufferers from leucocythæmia rarely present distinctive symptoms which can be associated with the malady until some time after its commencement, the most frequent error is to date its commencement late, and hence to assign to it too brief a duration.

The variations in the period during which there are distinctive symptoms are very great, and extend from a few weeks to many years. The most acute cases on record, in which the disease runs its course in a few weeks, are usually attended with great and rapid enlargement of the lymphatic glands and spleen, and apparently consist in an irritative process of such intensity that they can hardly be classed with ordinary cases of splenic leucocythæmia. The duration of the latter may be said to range from six months to seven years. One or two cases are on record in which the disease is said to have commenced and terminated in less than six months. Of these, one of the best authenticated is that recorded by Schreiber, in which death occurred in four months from the earliest symptom—profuse menorrhagia. But in this the hæmorrhagic tendency was well marked, and it is probable that the menstrual loss was the expression of a disease already developed. There are many cases on record in which the symptoms lasted for six months only. The majority of the cases terminate within three years of their commencement, but a few last longer, four or even five years. The following table represents the duration of the disease in sixty-three cases, in which the date of the earliest symptoms could be fixed with some accuracy.

	Males.	Females.	Total.
Less than 1 year	9	4	13
1—2 years	10	6	16
2—3 ,, ...	12	7	19
3—4 ,, ...	6	3	9
4—5 ,, ...	2	1	3
5 years and upwards	3	0	3
Total.	42	21	63

The average duration of cases of both sexes and all ages is nearly two years, the mean of the sixty-five cases being about twenty-three months.

Sex appears to exercise little influence on duration. The average duration of forty-one cases occurring in men was nearly twenty-four months, that of twenty cases occurring in women was not quite twenty-three months.

It may be doubted whether the number of cases available for comparison is sufficient to enable an accurate opinion to be formed concerning the influence of age on duration. As far as they go, the duration appears to be greatest at the period of life in which the disease is most frequent—between thirty and forty years. This is evident from the following table, exhibiting the relation of age to duration, in sixty-five cases of the primarily splenic form.

Age at commencement.	No. of cases.	Average duration.
10—20 years	11 cases	20 months.
20—30 ,,	18 ,,	22 ,,
30—40 ,,	18 ,,	29·8 ,,
40—50 ,,	12 ,,	19 ,,
50—60 ,,	5 ,,	27 ,,

The number is not sufficient to suggest any conclusion regarding the influence of sex on duration at the various ages.

Little can be said regarding the influence of individual symptoms on the duration. The cases in which enlargement of the lymphatic glands is an early symptom, run a course apparently rather more acute than others, but the number of such cases is not large. The size of the spleen appears to exercise no influence on the duration of the disease. Cases accompanied by pyrexia might be expected, from analogy with other diseases, to run a more rapid course than those in which the temperature is normal. This is probably true of cases in which pyrexia occurs early and is continuous. Many cases of long duration present considerable elevation of temperature towards the last. The cases in which there is no pyrexia frequently run a tardy course, and the exceptions to this, which are numerous, are due, in most instances, to the intercurrence of accidental causes of death, such as the cerebral hæmorrhage. No direct relation can be traced between the degree of leucocythæmia and the duration of the disease.

Many of the cases of longest duration presented before death an extreme increase in the relative excess of white corpuscles, and on the other hand, in some very acute cases the excess was comparatively slight.

CAUSES OF DEATH.—The course of leucocythæmia may end by a gradual weakening of the patient's strength, under the influence of the progressive anæmia or repeated loss of blood; by the direct effect of that loss of blood; or by the occurrence of independent local inflammations and other diseases.

Asthenia is a more common cause of death than any other single condition. Out of sixty cases in which the cause of death could be accurately determined, the end was due to simple asthenia in twelve. The gradual increase in the blood-state tends to increasing prostration, to increasing œdema, to increasing interference with the function of vital organs, until the patient sinks, commonly from cardiac failure. Consciousness may sometimes be retained to the last, sometimes obscured by the effects of the cerebral anæmia, which is a common effect of the state of the blood, and the weakness of the heart.

Loss of blood is a frequent cause of death, and was fatal in more than one quarter of the cases, in which the actual cause of death was noted (17 out of 60). The fatal hæmorrhage is usually external, sometimes internal, into some space into which a large extravasation can take place. The external hæmorrhage which is most frequently fatal is that from the nose—uncontrollable epistaxis, continuing for days, recurring whenever the plug is withdrawn from the nares. The next most frequent source of fatal hæmorrhage is the stomach. Hæmatemesis was the cause of death in four cases. Hæmorrhage from the bowels was fatal in three cases, in each there being also diarrhœa. Other internal extravasations causing death by loss of blood, have occurred into the peritoneal cavity and the cellular tissue. Considering how strong the hæmorrhagic tendency often is, it is remarkable that fatal hæmorrhage should so rarely result from traumatic causes. Splenotomy in several led to death by hæmorrhage; in one of the cases of peritoneal hæmorrhage, the cause was a puncture for paracentesis; and in one case death resulted from the loss of blood, which followed the extraction of a tooth and continued for two days. In cases in which hæmorrhage is not the immediate cause of death, it increases the anæmia and prostration, and accelerates the course of the disease and the fatal result.

Diarrhœa is another common cause of death, which in nine cases was attributed to this cause alone, without the occurrence of intestinal hæmorrhage.

Cerebral hæmorrhage was the cause of death in one-tenth of the cases—six out of sixty. Such a proportion is probably too large, and may be due to the circumstance that so striking a consequence of the disease led to a careful report of the case and its termination. Apoplectiform symptoms, due apparently to anæmia of the brain, preceded death in one case. In one case the cause of death was thrombosis in a cerebral sinus.

Lung diseases constitute another frequent cause of death. Pneumonia is of these the most important, and was the cause of death in six cases. Pleurisy and œdema of the lungs are very frequent towards the last, and sometimes have appeared to accelerate the end.

Rupture of the spleen was the cause of death in one case. In the remaining cases, death occurred from some independent intercurrent malady; septicæmia in one, varioloid in one, suppuration of vulval glands in one, and diphtheria in two cases.

PATHOLOGY.—The pathology of leucocythæmia is a subject which has received much attention, has occasioned much speculation, but is still obscure. On very few points is knowledge more precise, or hypothesis more definite, than when Virchow published his opinions on the nature of the affection a quarter of a century ago. This is largely due to our want of precise knowledge of the physiology of the blood, of the origin of the corpuscles, and their fate, and of the function of the organ which is chiefly affected in the disease. The theories which have been and are still advanced regarding its nature, are determined by the varying and different opinions held on these disputed points of physiology. In considering the pathology of leucocythæmia, the chief points to be discussed are:—

(1) The relation of normal to leucocytic blood.

(2) The normal history of the white corpuscles, and the functions of the organs which are diseased in leucocythæmia.

(3) The nature of the morbid change in the blood, and the relation to it of the pathological changes in the tissues.

(4) The relation of the morbid process to the causes of the disease.

(5) The origin of the variations in the symptoms.

Lastly, The occurrence of an analogous blood-state in other diseases.

(1) *The relation of normal to leucocytic blood.*—The condition in leucocythæmia is commonly stated in terms of comparison, *i.e.*, the relative numbers of white and red corpuscles. Under normal circumstances the proportion of white to red rarely exceeds 1 to 300 (1 to 347, or nearly ·3 per cent.) was fixed as the maximum by Moleschott. Frequently the proportion is far less, 1 to 500, or 1 to 1,000. The proportion is liable to considerable variations. It is increased after a meal, and is said to reach the maximum an hour after the meal.[1] Moleschott[2] found the proportion greater in children than in adults, and increased during menstruation. In observation on fifty persons he found the average numbers to be:—

In children	1	white to	226	red.
In men	1	,,	346	,,
In women	1	,,	389	,,
During the catamenia . .	1	,,	247	,,
Average of the whole . .	1	,,	347	,,

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[1] De Pury, Virchow's Archiv. vol. vii.

[2] Wiener Med. Wochenschrift, 1854, p. 113.

In leucocythæmia, as has been said, this proportion is greatly exceeded; the proportion of white to red being commonly greater than 1 to 5. A comparative statement of the relative numbers of the white and red does not, however, accurately express the condition of the blood, since there is always a double change, an absolute diminution in the number of the red, as well as an increase in the white corpuscles.

(2) *The normal history of the white corpuscles, and the normal function of the organs diseased.*—It is evident that, since all cells have a transient existence, such an excess of white cells may be due to their excessive formation, or to their undue persistence, in the blood; while the diminution in the red may be due to a deficiency in their formation, or to their undue destruction. A knowledge of the normal life-history of the corpuscles is therefore essential for the comprehension of their abnormal life-history in leucocythæmia.

What are the colourless corpuscles of normal blood, and what is their origin? With this problem is closely associated another: What is the relation of the blood to the normal functions of the organs which are found diseased in leucocythæmia? The two subjects must be considered together.

According to our present knowledge, the colourless corpuscles in the blood are formed from pre-existing cells of the same character (or from a protoplasmic network, according to Klein), mainly, if not exclusively, in the lymphatic organs of the body, from which they pass into the blood, directly, or indirectly by means of the lymphatic vessels. It is certain that leucocytes are thus formed in the lymphatic glands. It is probable that they are thus formed in the splenic follicles and in the follicular glands of the alimentary canal, and other similar structures, including the marrow of the bones, and it has been conjectured that they are also supplied by the thyroid and thymus glands.

The fact that colourless cells pass into the blood from the lymphatic glands is universally admitted, and is, indeed, hardly capable of dispute. It is probable, also, that corpuscles pass towards the blood from the lymphatic follicles of the intestines, since the lymphatic vessels contain corpuscles before reaching the mesenteric glands. That the spleen and the medulla of bones are normal sources of leucocytes rests also upon strong evidence. They are so frequently diseased when there is an excess of leucocytes in the blood, that the evidence as to their normal function is of considerable importance.

The opinion that the spleen in the normal state supplies leucocytes rests on the following facts. The round cells which constitute the chief cellular element of the spleen are identical in appearance and size with the colourless corpuscles of the blood. These cells are evidently in rapid process of formation in the spleen, either from pre-existing cells, or, according to Klein's recent researches[1] from the substance of the protoplasmic layers which limit the splenic and follicular spaces. They do not remain in the spleen, for they are not seen there in health in a state of degeneration, nor does the spleen exhibit that increase in bulk which would result from the retention within it of all the cells which are apparently being formed there. The structure of the organ is such as to facilitate the passage of these cells into the circulating blood. That such passage does occur is proved by the fact that blood which has passed through the spleen contains a much larger number of such cells than the blood which is going to the spleen. The number in the blood of the splenic vein is greater than that of the blood in the splenic artery.[1] On this important point there is a considerable amount of positive evidence. Hirt found the number to be 1 white to 2,260 red in the splenic artery, and 1 to 60 in the splenic vein. Funke, Malassez, and Picard, in their careful observations, came to a similar conclusion; but the latter believed that the difference was chiefly observed during digestion.

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[1] Quarterly Journal of Micros. Science, 1875, p. 363.

Other observers, especially Béclard, Lehmann, and Gray, came, however, to a different conclusion, and believed that the proportion of white corpuscles was reduced by the passage of the blood through the spleen. The discord between these observations is not, however, irreconcilable, or incompatible with the assumed function, as will be shown immediately. There is reason also to believe that red blood corpuscles are formed in the spleen. It was maintained, in the last century, by Hewson, and the opinion has been widely accepted by physiologists, that lymphoid corpuscles are converted into red corpuscles in the spleen. It was suggested by Virchow (and also in effect by Bennett) that a large number of the splenic cells, in passing into the blood, undergo immediate development into red corpuscles. Considerable evidence has accumulated in support of this theory. The blood, when it leaves the spleen, is absolutely richer in red corpuscles than the blood which enters the spleen. This was ascertained by Malassez and Picard,[2] who counted the number of corpuscles per cubic millimeter in the blood of the splenic artery and vein respectively. Their numerical observation was corroborated by the fact that the capacity of the blood of the splenic vein for absorbing oxygen was greater than that of the splenic artery. They found also paralysis of the spleen, by section of the nerves, which increases the circulation through it, increases the excess of corpuscles and the oxidizability of the splenic venous blood,[3] while the iron in the spleen becomes lessened. Moreover, Funke found in the blood of the splenic vein the same transitional forms which Kolliker noted in the spleen, and also many corpuscles which were smaller and rounder than the others, had less tendency to run together, and resisted water to a greater degree.

It is evident, then, that the number of white corpuscles in the splenic vein will depend not only on the number formed in the spleen and passing into the blood, but on the proportion which is transformed into red corpuscles. Some of the younger lymph cells brought to the spleen by the blood may undergo the same transformation. The proportion of cells thus converted will vary under different conditions and at different times. The greater it is, the smaller will be the proportion of white corpuscles in the splenic vein. Thus the opposing results may be reconciled and harmonised with the other ascertained facts.

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[1] Funke, Henle's Zeitschrift, 1851, p. 172, quoted by Bennett.

[2] Comptes Rendues de l'Acad. de Sciences, Aug. 12, 1876.

[3] This is, as the experimenters note, the reverse effect to that which is obtained with other glands.

Another function has been attributed to the spleen, that of destroying red corpuscles. It is inferred from the occurrence in the spleen of masses of degenerating corpuscles, and from the fact that the spleen contains more iron than corresponds to an equal quantity of blood. The same fact has indirectly been assumed from the circumstance that excision of the spleen commonly causes the bile to be devoid of pigment. It is assumed that the corpuscles destroyed in the spleen furnish the pigment of bile. The conclusion from this fact cannot be admitted to have much weight. The mechanism of their destruction is uncertain. It has been suggested that the lymphoid corpuscles, amœba-like, enclose and destroy them, but Klein concludes, from his researches, that the destruction is effected rather by the inter-cellular splenic protoplasm.

Excision of the spleen has been expected to throw considerable light on its functions, and indirectly on the effects of its disease. The results of the experiment have been, on the whole, disappointing. The clearest fact, one which was established two centuries ago by Oldenburg,[1] and corroborated in the last century by Hewson[2] is, that it is not an organ essential to life or health; that animals which recover from the operation of its removal live on, quite well, without it. Increased appetite has been observed by some experimenters, but it does not appear to be an invariable symptom,[3] and the explanation given of it by Schiff is not very satisfactory. Hypertrophy of the mesenteric glands has been noted after a time,[4] and Ludwig found, in one case, that the enlarged glands possessed a vascular network similar to that of the spleen. Mosler failed, however, to discover anything of the kind, but in the medulla of the bones of a dog. the spleen of which had been extirpated ten months before, Neumann found marked changes, an enormous increase of lymphoid cells and intermediate forms, compared with the normal medulla of the dog, and with that of dogs a few days or weeks after the extirpation of the spleen.

Bardleben noted watery effusion into the cavities, and in Ludwig's case the dog died from hæmorrhage. The latter fact suggests an alteration in the blood, and although none was found by Bardleben, the more careful observations of Mosler, by Welcker's method, have shown that the blood, a month after extirpation, was notably paler; the number of white corpuscles was twenty times less than in health; and the red corpuscles much fewer; the diminution continued, being still more marked a few months after extirpation. These facts afford strong corroboration of the function of the spleen in the formation of white and red corpuscles, which has been inferred from microscopical investigations of its normal structure. The application of these facts to the explanation of splenic leucocythæmia will be considered immediately.

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[1] Philosophical Trans., vol. ii. 1667, p. 521. Oldenburg excised the spleen of a bitch which afterwards bred.

[2] Med. and Philos. Comment. 1775, vol. iii. p. 87; republished in the Sydenham Society's edition of Hewson's works.

[3] Mosler, Lussana.

[4] Czermak, Tiedemann and Gmelin, Hyrtl, Ludwig.

Recent observations on the structure and constituents of the medulla of bone, especially those of E. Neumann, afford strong ground for attributing to it a function similar to that of the spleen in the formation of blood. The vascular arrangement of the tissue is peculiar. The transition from capillaries to veins is very gradual, the veins are large in size, and possess very thin walls.[1] It has been said, indeed, by Hoyer,[2] that the blood passes through spaces, devoid of distinct walls, into which the capillaries open, and from which the veins bring the blood. Such a structure could not be observed by other observers. It is clear, however, that the arrangement of the vessels is such that the passage of the blood through the medulla is slow, just as it is through the spleen. Abundant lymphoid cells exist in the normal medulla, and appear to be in process of multiplication. Numerous cell-forms have been observed, first by Neumann, and afterwards by Bizzozero, Morat, and others, which appear to present every stage of development from lymphoid cells into red blood-corpuscles. The forms correspond closely with the cells from which blood corpuscles are seen in process of development in the embryo. The nucleus, according to Bizzozero, enlarges, acquires a yellowish red colour, and the cell presents the appearance of a red blood-corpuscle surrounded by a thin layer of protoplasm. Within the capillaries may be seen the lymphoid cells, and also the intermediate forms, in large numbers (Neumann). It is assumed, therefore, that leucocytes are produced outside the vessels, pass into the capillaries, and in the slowly moving blood undergo development into red globules. Bizzozero believes, moreover, that a destruction of red corpuscles also occurs on the medulla.

What are we to conclude is the fate of the pale blood-corpuscles? It appears probable that a few of them persist and perish, as such, in the blood.[3] Many of them pass out of the blood into the tissues, and there subserve processes of growth in a manner at present ill-understood. They also, without doubt, leave the vessels wherever inflammation is going on, and appear in the inflammatory products as pus corpuscles, of which they constitute an important, perhaps the only source. Other white blood-corpuscles probably divide and give rise to similar cells. It is uncertain, however, whether this process goes on outside the lymphatic tissues. Within the glands and follicles of lymphoid tissue, such a multiplication is probably always going on, but it is not yet proved that the lymphoid corpuscles, which have left the lymphoid tissue, and have passed into the blood, undergo such division and multiplication. Lastly, it is now believed that a large number of white corpuscles are transformed into red ones, and that this transformation constitutes the only source of the red globules, and a chief destination of the colourless cells. There is reason to believe that the change takes place mainly in the tissues of certain organs, especially the spleen and the medulla of bone. The precise character of the change which the corpuscles undergo is still a disputed point, and it is not essential for the discussion of the pathology of leucocythæmia.

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[1] E. Neumann, Centralblatt, 1868, p. 885; Archiv. der Heilkunde, 1869, x. p. 68; Hofmann and Langerhans, Virchow's Arch. vol. xlviii. p. 309.

[2] Hoyer, Centralblatt, 1869. Nos. 16 and 17.

[3] Virchow, in his Archiv. i. p. 144, and p. 593, pointed out that the degeneration they occasionally present is evidence of this.

It was long ago urged by Virchow that all the colourless corpuscles are not capable of this transformation, since some may be seen undergoing fatty degeneration, and others which have suffered such a change that their nucleus is, or can be, broken up, and thus they resemble in all particulars, while circulating in the blood, the characters assumed by those which have passed out of the vessels and are called pus cells. He believed that the transformation occurs only at an early epoch in the development of the cell, before it reaches the circulating blood, and that when it has reached the latter it has become a "simple" not a "specific" cell, and its further transformation into a red globule is impossible. Virchow regarded these colourless corpuscles thus as a "relatively superfluous constituent of the blood, a residuum." We know more now of the purpose served by these corpuscles; that they have a function of great importance by their power of wandering into the tissues. Moreover, there is no additional reason to believe that many circulating corpuscles are not capable of transformation. But, with these reservations, there are additional facts which support Virchow's view, and it is a theory of much importance in connection with the pathology of leucocythæmia.

(3) *The nature of the morbid Change in the Blood, and the relation to it of the Pathological Changes in the Tissues.*—The two conditions must be considered to some extent together, and this is especially the case as regards splenic change.

The first question for consideration is, whether the excess of white corpuscles present in the blood in leucocythæmia depends on an increased production, on a diminished transformation into red, or on both these conditions.

It is probable, in the first place, that there is a diminished transformation. Whenever the white corpuscles are increased, the red are diminished in number. The greater the increase in the white, the greater is the decrease in the red. It is of course conceivable that this relation might be an indirect one, that the changes which accompany the increase in the white cells might lead to a great destruction of the red. But such continuous destruction of the red could not occur without being revealed by indications which are absent in leucocythæmia. It is therefore certain that, although possibly the destruction of the red globules may be greater than in health, such destruction is not the main cause of the diminution in their number, but that this is chiefly due to a decreased formation. But since, as far as we know, the red globules are formed only by the transformation of lymphoid cells, the diminished formation of the red is evidently due to either diminished formation of the lymphoid cells or to their diminished transformation. But the number in the blood is hardly compatible with a diminution in their production, and there must therefore be a defect in the transformation of the cells into red corpuscles.

This view is corroborated by the circumstance that a very large proportion of the pale corpuscles in the blood present degenerative changes which may be considered as incompatible with their further transformation. Some of them are seen to have undergone actual fatty degeneration. In most of the others acetic acid renders a double, tripartite, or horse-shoe nucleus visible (see Fig. 2). It was maintained by Virchow that this effect is to be taken as the result of degeneration of the nucleus and as evidence of the cessation of the active life of the cell. It was long ago pointed out by Donné[1] that these white corpuscles are indistinguishable from pus corpuscles. The discovery of diapedesis renders it probable that they are not only indistinguishable but identical.[2]

Is there an increased formation of leucocytes as well as a diminished metamorphosis? The answer is difficult to give. On the theory of defective transformation of white into red, the blood state may be amply accounted for. Where the disease is manifested only as a change in the blood without any alteration in the structure of the lymphatic organs (as in Dr. Lloyd Robert's case),[3] or such change is trifling (as in the case of Dr. Moxon),[4] it seems to be a sufficient explanation of the condition. But in most cases of leucocythæmia there is an extensive overgrowth of lymphatic tissue, with a corresponding increase in the lymphoid cells contained in the tissue. Taking this overgrowth in connection with the fact of the leucocytal excess in the blood, the conclusion that there is an excessive formation of leucocytes has usually been accepted as a necessary one.

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[1] Cours de Microscopie, 1844, p. 196.

[2] Vidal, in his conclusions (De la Leucocythémie Splenique, Gaz. Hebd. 1856, p. 253) regarding the relation of white corpuscles and pus cells, certainly anticipated more recent views. "Les globules blancs de sang, les globules de pus, de mucus, ne sont qu'une même espèce de globules :—Ce qu'on a décrit comme globule de pus et du mucus n'est pas autre chose que le globule blanc de sang ; ainsi les globules blancs du sang seraient un des elements constitutifs du pus, ce qui nous rapprocherait de l'opinion des anciens sur la question de la pyogénie ; et nous ne serions pas etonné si le microscope venait demontrer un jour que tous les elements du pus son des elements homœomorphes, contrairement à la doctrine le plus géneralement accepté à notre epoque."

[3] Brit. Med. Journal, Nov. 27, 1869.

[4] Clin. Soc. Trans. 1877.

The problem is in some respects a simpler one as regards the lymphatic glands than as regards the spleen. In the rare cases in which the lymphatic glands are the seat of simple soft enlargement, due to an excessive quantity of tissue differing but slightly from the normal tissue of the glands, and in which the blood contains a considerable excess of colourless corpuscles resembling the cells of the enlarged glands in every particular, the evidence is very strong in favour of an increased formation and increased supply to the blood from the diseased glands. Even here the possibility of diminished transformation cannot, by any facts at present at our disposal, be entirely excluded. It is, as just remarked, possible that the young lymphatic cells, after reaching the blood, may, in it, or in certain organs, as the spleen and marrow, undergo the transformation into red globules. That this is the case is probable from the fact that a diminution in the number of red globules accompanies glandular diseases in which, apparently, the supply of cells to the blood is arrested by changes in the structure of the glands. But, on the other hand, a leucocythæmia may sometimes result from a local growth, and may be out of proportion to the anæmia, as in the case of Lücke's, and in one under my own observation, in which seventy white corpuscles were present in every field of blood, from a patient with a local cervical lymphadenoma. This is evidence that, under some circumstances at least, the supply to the blood from the lymphatic glands is increased, and renders it probable that in soft glandular enlargements, in which a path to the blood is demonstrable by injection, this is often the case.

Regarding the spleen, the problem is more complex. The spleen in leucocythæmia undergoes great enlargement, which is due, mainly, to an increase in the splenic pulp. The splenic follicles consist of adenoid tissue with large numbers of lymphoid cells in process of formation, and it is believed that these cells, formed in the follicles, pass from them into the meshes of the splenic pulp (where, perhaps, other similar cells are formed), and are there transformed into red corpuscles and added to the blood. The splenic pulp in leucocythæmia is increased in quantity, apparently by the growth of the lymphoid tissue in it. There is an enormous increase in the lymphoid corpuscles which it contains. This increase has suggested to most pathologists the occurrence of a rapid formation of corpuscles in it. But the suggestion occurs, Is not the pathological condition accounted for by the mere retention in the splenic pulp of large numbers of white corpuscles? That such retention and accumulation does actually occur, can hardly be doubted. It must be the chief process in anæmia splenica, in which there is a simple "hypertrophy" of the spleen similar to that which occurs in leucocythæmia, a great deficiency of red corpuscles in the blood, but no excess of white, although numbers of similar cells are collected in the splenic pulp. But such accumulation cannot, in the spleen of either anæmia or leucocythæmia, be the whole process. The enormous increase in the bulk of the spleen, growth and development of the tissue elements of which it consists. The splenic corpuscles under consideration must participate in the change. especially if, as Klein believes, they are derived directly from the protoplasm of the stroma. Their tendency of development may be so affected that they no longer change to red globules, but remain as lymphoid cells, and in the blood persist as white blood corpuscles. The second hypothesis is that the leucocytes undergo their peculiar transformation in certain organs, under some influence, perhaps chemical, exerted upon them by the constitutents of those organs. There is reason to believe that the change occurs in the spleen and medulla of bone to a greater extent than in the blood, since intermediate forms are found more abundantly in those organs than in the blood at large. Corpuscles which come from the lymphatics, must, if transformed at all, be transformed after entering the blood, and that they are so changed is probable from the profound anæmia induced by general disease of the glands, unattended by splenic change. That the vital properties of the corpuscles, their tendencies of development, are changed by environment, is probable from the fact that white corpuscles which have left the blood, and escaped as pus, rapidly present evidences of degeneration and never of metamorphosis into a red globule. If this metamorphosis depends on the influence exerted on the corpuscles by certain organs, or by the blood, that influence may be so changed by disease of those organs, or by a morbid state of the blood, that the metamorphosis is hindered, is attained by but few corpuscles, and hence there result a progressive deficiency of red and an excess of white corpuscles.

It has been urged that this defective transformation, due to a change in the blood, constitutes the whole initial lesion in leucocythæmia: that the splenic enlargement is due only to the accumulation within it of leucocytes, which, from defective transformation, exist in large quantities in the blood, and are arrested in the splenic tissue. This view was originally suggested by Griesinger[1] and it has been adopted by Cayley, Kottmann, Colin, and others, and has been recently urged by Biesiadecki, who bases it on the following considerations: (1) that the anatomical change in the spleen does not suggest any excess of formation; (2) that there is an atrophy, instead of an hypertrophy, of the parenchyma of the organ, the chief change being an accumulation of cells in the perivascular tissue, in which he has found pigment massed in the melanotic spleen of ague; (3) that the corpuscles present degenerative changes which show that no further transformation can take place; and (4) that the absence of any ill-effects in extirpation of the spleen is hardly compatible with the view which leucocythæmia suggests of its functional importance as a blood-making organ. That an essential condition in leucocythæmia is defective transformation of white into red, that such defective transformation may be the only morbid condition in some cases, and that such accumulation in the spleen may occur, has been urged in the preceding pages, and was, in effect, suggested long ago by Virchow. But the enlargement of the spleen in leucocythæmia can scarcely be explained by the accumulation within it of cells brought by the blood. Many cases are on record which show that a splenic enlargement may precede any excess of leucocytes in the blood,[1] and make it probable that this is, in most cases, the order of events. The most distinct causal influence (ague) is one which acts directly on the spleen, and, further, a primary lesion of the spleen, caused by section of its nerves, may produce a considerable excess of leucocytes in the blood.[2] The view that there is an atrophy of the splenic tissue is not in harmony with the conclusion of most pathologists, who have uniformly asserted that the splenic follicles become indistinct, but the tissue elements of the pulp undergo a vast increase and cause the enlargement of the organ. With this my own observations entirely accord. Were there only an accumulation within the capsule of the spleen of fifty times the weight of leucocytes, it is needless to say that no splenic tissue or consistence would be recognisable. Moreover, there is evidence that other lymphatic tissues may participate in the initial change and even be alone affected. Lastly, the effects of excision of an organ are of no value as evidence of the effects of its disordered function. A lost influence may be replaced by that of other organs, while the excess or perversion of it may entail grave and enduring consequences.

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[1] Virchow's Archiv. Bd. v. p. 391.

The relation to the blood state of the changes in the other lymphatic tissues and in the organs must be considered in further detail.

Glands.—The glandular changes in leucocythæmia must be regarded in a somewhat different light, according as they occur at the beginning of, or late in the course of, the disease. In the rare cases in which simultaneous enlargement of the glands and the spleen occurs, and in which the spleen is changed as in primary splenic leucocythæmia, the glands are the seat of a primary lymphatic overgrowth, which may precede the development of the hæmic change to which it probably in some degree contributes. These cases are intermediate between splenic leucocythæmia and lymphadenosis, or, rather, present a combination of the two morbid states, and belong in their pathology to both. Their aspect, as they relate to the lymphatic glands, will be considered more fully in the next article. It may be here mentioned, however, that they afford distinct evidence of the origin of the disease in an affection of the lymphatic tissues and not of the blood.

In another set of cases the glandular enlargement occurs late in the disease, after the leucocythæmic state has been developed, and has existed for some time. Two views have been held regarding the pathology of this condition. Virchow regarded it as a lymphoid growth, analogous to that which sometimes initiates the disease, but occurring secondarily to the splenic affection, and indicating a stage of generalisation of the disease. It has, on the other hand, been urged, originally by Dr. Wilks, that such gland changes are due simply to the accumulation in the glands of leucocytes brought to them by the circulating blood, or, according to Dr. Moxon, brought from the tissues by the lymphatics. But this theory rests upon no direct evidence, and is opposed by certain facts. In the first place, there is no relation between the occurrence of these glandular changes and the degree of leucocythæmia. If the theory were true, the greater the leucocytal excess, the more constantly should glandular enlargements occur, and the greater should be their degree. But no such relation can be observed. Many of the cases in which the excess of white corpuscles was extreme, and in which even growths existed in the organs, presented no glandular enlargement; while in others, in which the glands were affected, the excess of leucocytes was comparatively slight, and the glandular enlargement occurred early in the disease. The independence of the two conditions goes farther, since the glands may be the seat of general enlargement with an overgrowth of splenic pulp, without any excess of leucocytes in the blood.[1] Thus the circumstances suggest strongly that the glandular affection and blood change, are, at least to a large extent, independent, and that the glandular affection depends, as Virchow supposed, on a lymphatic growth. The same opinion is suggested by the microscopical structure of the enlarged glands. When soft, as they commonly are, they present large quantities of lymphoid cells, but these are, as a rule, smaller than those of the spleen. Moreover, there may be an increase in the retiform tissue as well as of the cells, so that the gland may have a tolerably firm consistence. It is not denied that some accumulation of cells may take place, but it does not seem probable that the enlargement is primarily or mainly due to such accumulation.

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[1] Two cases are recorded by Bennett, Leucocythæmia, p. 128, Case 36; and Day, Clinical Histories and Commentaries.

[2] Von Tarchanoff, Pflüger's Archiv. 1874, Bd. viii. p. 97.

Medulla of Bone.—The frequency with which the bone-changes, already described, are met with in cases of leucocythæmia, shows that the association cannot be an accidental one. The evidence, already mentioned, that this structure has a blood-forming function, led Neumann to compare the disease of the medulla to that of the spleen. Further observations have shown that the change may, with greater accuracy, be compared to that in the lymphatic glands. It seems to occur, usually, as a secondary affection, late in the case. The great increase in the number of lymphoid cells, and the resulting changes in structure, are comparable to the changes in the lymphatic glands. Like the latter, also, the medullary change bears no relation to the amount of blood-change, or to the degree of splenic enlargement, and may occur without any excess of leucocytes in the blood (in lymphadenosis and pernicious anæmia), and be absent when there is extreme leucocythæmia. The same opinions have been expressed regarding its nature. It has been looked upon by Neumann, Mosler, &c., as an independent lymphatic overgrowth, with the same effects upon the blood. By others, as by Dr. Moxon, it has been regarded as a purely secondary change, the consequence of the accumulation within the structure of leucocytes brought to it by the blood. The correctness of the latter view may be doubted from the apparent independence of the marrow changes and leucocytal excess, in occurrence and degree. It is probable, therefore, that it depends on a simultaneous lymphatic overgrowth.

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[1] For examples of this see the next article.

There is some reason to believe that this change may be the primary alteration in leucocythæmia. Thus in a case recorded by Mosler,[1] in which leucocythæmia was associated with much enlargement of the spleen, there was an extreme alteration of the medulla, the iliac bone, ribs, and especially the sternum, being much swollen and soft. Sternal pain preceded for four years the first symptoms which could be attributed to the splenic enlargement. It has even been thought that there may be a pure myelogenic form of leucocythæmia, a general bone change being the only pathological alteration. The evidence is at present insufficient to establish this, but there are facts which show that a primary bone disease may be associated with an excess of pale corpuscles in the blood. In a case recorded by Waldeyer[2] (on which, perhaps, undue stress has been laid as evidence of a purely myelogenic form of leucocythæmia), a local osteomyelitis of one tibia, followed by pyæmia, was associated with such an excess of leucocytes in the blood, that 150 were present in the field of the microscope. But the spleen was enlarged to eight and a half inches in length (partly from embolism), and the right iliac glands and the intestinal glands were also somewhat enlarged. A case in which an osteomyelitis, following an amputation of the thigh, was associated with a large excess of pale corpuscles in the blood, was recorded by Mursick.[3] Moreover, a local growth in bone seems capable of entailing a state of leucocythæmia just as did an axillary lymphadenoma in the case described by Lücke.[4] Heschl recorded a case in which a mass of growth in the femur, the size of a child's head, apparently arising in consequence of a blow, was associated with enlargement of the inguinal and mesenteric glands, and a great excess of leucocyte-like cells in the blood. The clots in the heart were greyish red, and in some blood from a small vein the proportion of white to red corpuscles was estimated with the microscope to be as one to three.[5]

Intestinal Changes.—The enlargement of the lymphatic follicles of the intestine occurs, as a rule, late in the disease, and has been regarded as part of the generalisation of the affection. The increase is due, in most cases at least, to an additional lymphoid growth, often extending far beyond the original limits of the follicle. These growths occur, as does the enlargement of the lymphatic glands, independently of the degree of leucocythæmia, and independently even of its existence. They are seen, for instance, in lymphadenosis; and Cohnheim has described[1] a remarkable case in which the spleen was enlarged as in leucocythæmia, the intestinal follicles were enlarged, and minute lymphoid growths existed in various organs, without any excess of white corpuscles in the blood. It is clear, therefore, that the intestinal change cannot be due wholly to the excess of leucocytes, as has been asserted. It is, of course, possible that the excess may increase the follicular growth, although there is not at present any direct evidence to show that such is the case.

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[1] Berlin Kl. Wochenschrift, 1876, pp. 49–51.

[2] Virchow's Archiv. Bd. lii.

[3] New York Med. Record, 1868, March.

[4] Virchow's Archiv. vol. xxxv. 1866, p. 524.

[5] Virchow's Archiv. 1855, vol. viii. p. 355.

The intestinal growth may supply white corpuscles to the blood and increase the degree of leucocythæmia. It has been thought that it may even be alone a source of a large excess, as in a case recorded by Behier, as "intestinal leucocythæmia."[2] The patient was a man, aged 25, who suffered from weakness, pallor, dyspnœa, and the blood presented an equal number of white and red corpuscles. Post-mortem, the spleen was scarcely enlarged, the glands everywhere normal, and Peyer's patches were so thickened as to project three millimeters above the surface of the intestine. A somewhat analogous case in a child has been described by Virchow.[3]

Visceral Changes.—The pathology of the other visceral changes in leucocythæmia has received a different explanation in different cases. The occurrence of aggregations of lymphoid cells, among which, in some instances, a distinct stroma of retiform tissue could be traced, led Virchow to regard them as lymphatic growths, arising in the lymphatic interstices of the tissues and organs, and an indication of the generalisation of the disease. In other cases, the conspicuous distension of the capillaries with white blood-corpuscles, and the occurrence of aggregations of white corpuscles in the immediate vicinity of vessels, has suggested the explanation that these are merely local aggregations of extravasated leucocytes, escaped from the vessels by rupture or diapedesis.

It can scarcely be doubted that each of these views is true, and that the organic changes have this double origin. The lymphatic structure of some of the new formations is very distinct, and the stroma evidently different from that of the organ in which they occur. The size they occasionally attain precludes any explanation but that of a process of growth. On the other hand, the obstruction of capillaries by leucocytes is often very distinct, and the position of aggregations of leucocytes adjacent to the vessels renders the view that they have escaped at the spot a probable one. The extent to which the two processes of growth and extravasation are combined to produce the pathological changes probably varies in different cases, and some morbid appearances may be interpreted almost as well on either theory.

Two circumstances must ever be borne in mind in drawing any conclusion from the position or characters of an apparent growth or extravasation. First, that the vicinity of vessels is that in which the lymphatic tissue is most highly developed and in which other lymphoid growths, by preference, occur. The position on a vessel must not therefore be taken as conclusive proof of origin by extravasation. Secondly, it is by no means improbable that an extravasation of leucocytes, or an aggregation of wandering leucocytes, may constitute the nucleus of a lymphoid growth. The cells may possess, in the constitutional state, a somewhat infective property, by which they may multiply, and induce the development of a stroma of retiform tissue, in their points of aggregation. By a similar means, lymphoid growths in progress may be reinforced. This is the theory of Virchow. If it be true, it has been thought that leucocythæmia might be communicable by inoculation. Mosler has failed to produce the disease by the injection of leucocythæmic blood.

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[1] Virchow's Archiv. vol. xxxiii. 1865, p. 451.

[2] L'Union Médicale, August, 1869, Nos. 99 and 100.

[3] Gesammt. Abhand. p. 199.

The probable origin of the various symptoms of leucocythæmia has been considered, as far as our knowledge extends, in the description of those symptoms.

Relation to Anæmia Splenica.—The relation of splenic leucocythæmia to splenic anæmia has been incidentally discussed in the consideration of the origin of the excess of pale corpuscles. The passage of the corpuscles into the blood, or their retention where formed, is the only difference which a pathological scrutiny can suggest. It is possible that in the anæmic form the production of leucocytes is less than in the leucocythæmic form, and that an increased formation, or an accumulation out of proportion to the amount of stroma, may constitute the transition of the one form into the other—a transition which sometimes occurs early and sometimes late in the course of the disease. It is remarkable how closely the pathological changes and even the symptoms of leucocythæmia are paralleled by the cases of simple splenic anæmia. In Cohnheim's case, already mentioned, the remarkable growths in glands, liver, spleen, kidneys, tonsils, and intestines were all secondary in time to a splenic enlargement, and the patient had had ague two years before. In two other cases, one with a glandular enlargement and the other with enlargement of the liver, there was similar evidence of paludal poison. The symptoms present many similar features, and the tendency to hæmorrhage may be marked as in leucocythæmia. In Cohnheim's case there was daily epistaxis; and in a case recorded by Lissauer,[1] excessive hæmorrhage on the extraction of a tooth was the first symptom. It is only by the secondary consequences of the leucocytal excess that the morbid states can be separated in their symptoms.

(4) *The relation of the morbid process to the causes of the disease.*—Of the relation of leucocythæmia to its ultimate causes we know very little. It is only by the study of cases early in their course that more can be learned on this point. The causes to which a relation

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[1] Berlin Kl. Wochenschrift, 1865, ii. p. 403.

can be most clearly traced, appear to act primarily on the lymphatic organs, in which the chief anatomical changes are found. Intermittent fever, for instance, leads to acute and chronic enlargement of the spleen, and this precedes the change in the blood. The mechanism by which the enlargement of the spleen is produced is unknown. It appears, however, that the acute enlargement is attended with a diminished supply of white corpuscles to the blood, *i.e.*, an arrest of their formation, or a retention of those formed within the spleen: probably the latter. In the early acute enlargement there is a great increase of the amount of blood in the spleen. There must, therefore, be a vaso-motor paralysis. But simple paralysis of the spleen leads to an increased production, and an increased supply to the blood, of splenic cells. Von Tarchanoff[1] found that the division of the splenic nerves caused enlargement which was accompanied by the presence of from 40 to 70 white corpuscles in each field on the second and fourth day, instead of the normal 10 to 15. The excess and the splenic tumour lessened together. In the congestion of intermittent, it would seem that whether there is or is not an increased formation of leucocytes, there is a diminished supply to the blood. This number is smaller than normal during intermittent, and in cases of cachexia with simple splenic enlargement.[2] The retention of the cells probably gives rise to the persistent enlargement which occurs in many cases, and, at least in part, to the anæmia which results. The passage of some of these cells into the blood in undue quantity appears to be one element in the origin of leucocythæmia.

But intermittents exercise an influence on another blood-forming structure, the marrow of bones. Browicz[3] has found that after repeated intermittent the marrow loses much of its fat, and the tissue assumes an embryonal character. Round, nucleated cells replace the fat cells, and pigment accumulates in its structure.

The blood itself is apparently directly affected. In recent cases it contains a large quantity of free pigment. After a time, the pigment disappears from the circulating blood and accumulates in certain organs; in the splenic parenchyma (not in the splenic follicles), in the medulla, and in the interacinal tissue of the liver. The pigment is supposed to arise from the destruction of red blood-corpuscles.

Another important relation of leucocythæmia is to the puerperal state. In several cases the association has appeared clear. It derives significance from the fact that during pregnancy, and especially after delivery, there may be a marked tendency to the occurrence of lymphatic changes. During pregnancy, there may be, as will be mentioned presently, an excess of leucocytes in the blood, and in febrile attacks after delivery the excess may be very great. Some remarkable cases are on record in which, after delivery, an acute swelling of spleen and glands, originating then, or during the preceding pregnancy, was accompanied by leucocythæmia, and ran a rapid and fatal course.[1] Of the possible mechanism of these relations we are still ignorant.

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[1] Pflüger's Arch. 1874.

[2] Kelsch, Arch. de Physiologie, 1876, p. 490.

[3] Virchow's Jahresbericht, 1876, i. 275.

In other cases in which no etiological relation could be traced, we must assume the existence of a primary change in the lymphatic tissues, especially in the spleen. Why, in some cases the glands, in others the spleen, in others both, should be affected, we have no evidence. It is probable that the spleen is the seat of more frequent and considerable variations in its vascular state, due to the influence on it of the state of the alimentary canal, than any other collection of lymphatic tissue, and that the variations may lead to irritative changes, initiating those which, in a certain constitutional state, result in leucocythæmia. The existence of a widely spread predisposition in some cases, is seen in the simultaneous affection of spleen and glands. In an instructive case recorded by Vogel,[2] there was evidence of this double predisposition, leading, under irritation, to a transient glandular and persistent splenic enlargement. Glandular swellings in the neck and groin preceded the splenic enlargement. The former seemed to be due to an angina. They subsided; then the spleen enlarged, then the cervical glands again enlarged, and suppurated and healed, but the splenic enlargement persisted.

(5) *The origin of the Variations in Symptoms.*—There are many characters of leucocythæmia which we are at present unable satisfactorily to explain. One of these is the remarkable variation in the symptoms, blood-state and splenic and glandular enlargements, which some cases present. The variation in the number of white corpuscles in the blood is occasionally very great. It may be in part due to variations in the number of white corpuscles which undergo transformation into red. It is probable that the metamorphosis is largely influenced by the varying general condition of vitality. This may be one explanation of the excess occasionally observed in acute febrile diseases. The variations in the pyrexia and general state in leucocythæmia may well be attended with a similar result. It is probable, again, that a contraction of the spleen, by whatever induced, may be accompanied by an expulsion of corpuscles into the blood. This has been established with regard to the normal spleen and the enlarged spleen of malaria. In leucocythæmia, the variations in the size of the spleen are of frequent occurrence, and may induce consequent variations in the state of the blood. Hæmorrhage again, which, in the normal state, causes a temporary increase in the number of pale corpuscles, may have a similar effect in leucocythæmia. A large hæmorrhage, external or internal, which lessens very much the volume of the blood, may cause a remarkable diminution in the size of the spleen.[3]

A sudden increase in a local growth may be attended by a great diminution in the degree of leucocythæmia, and even in the size of the spleen and lymphatic glands. Cases illustrating this have been recorded by Mosler[1] and Shingleton Smith.[2] The explanation probably is that such growth is due, in part at least, to a vast accumulation within it of the circulating leucocytes, sufficient to relieve the blood and drain the spleen and glands.

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[1] Paterson, Edin. Med. Journal, June, 1870.

[2] Virch. Arch. Bd. iii.

[3] Vidal, Gaz. Hebd. 1859, p. 167.

The rapidity with which great changes in the state of the blood may occur is very remarkable. This is seen in some cases which cannot be regarded as splenic leucocythæmia. Litten[3] has described a case of acute anæmia of three weeks duration, in an intemperate man, in whom, till within the last four days of life, there was no marked increase in the number of white corpuscles, and during that time the increase was so rapid that on the day of death the proportion of white to red was as one to four. A case in which there was almost constant permanent excess and still more rapid and remarkable variation, has been published by Dr. Goodhart.[4] Within a day or two, the blood-state varied from no excess of white to almost equal numbers of white and red, and the variation recurred every few days. At the post-mortem, in which simple enlargement of the spleen to 17oz., glandular enlargement, and lymphoid growths in liver and kidneys were found, nothing was discovered to explain the alternation. Remarkable variations in the number were present in a case described by Dr. Day as "alternating leucocythæmia," but it is probable that in this case they must be ascribed in part, if not entirely, to the frequent hæmorrhage, and the case can scarcely be classed with those of splenic leucocythæmia.

(6) *The occurrence of Leucocytal Excess in other diseases.*—In many morbid states an excess of leucocytes may be present in the blood. The number may vary from the normal two or three, per quarter-inch field of the microscope, to ten, twenty, thirty, or fifty, but rarely exceeds the latter in any but splenic diseases.

Hæmorrhages may cause a temporary increase in the proportion of leucocytes in the circulating blood. The first effect of a hæmorrhage is, of course, to diminish the corpuscles and the serum, in proportion to one another. It has been found by Mr. W. S. Tuke, in some observations he has kindly made for me with the hæmacytometer, that, in man, immediately after a venæsection, the blood exhibits nearly the same number of white and red corpuscles as existed before the operation. There is, however, a rapid passage of liquid from the tissues to the blood, and hence the number of corpuscles per cubic millimeter of blood rapidly falls and attains its minimum usually from half an hour to an hour after the hæmorrhage. It then begins to rise, apparently from an increased supply of cells to the blood. This rise is often accompanied by a considerable excess of pale corpuscles, which is the greater, the larger the hæmorrhage,—is greater in certain constitutional conditions than in others, and is far greater in some animals than in man. In the horse, for instance, it is said that the numbers of red and white cells may be rendered equal by a large hæmorrhage. There is little or no evidence as to the mechanism by which this is effected. Probably the defective number of corpuscles may lead to the rapid passage of leucocytes into the blood, and their removal into the circulation before their metamorphosis into red ones; and the excess may disappear from the gradual conversion of these cells into red globules. It is not difficult to understand that loss of blood may lead to the passage of pale cells into the blood in large numbers, since the diminution in the size of the spleen from a diminution in the amount of blood in it, may lead, by the contraction of its elastic capsule, to the expulsion of free leucocytes from the meshes of the splenic tissue and follicles, an effect analogous to that which is produced when contraction is caused by the application of electricity.

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[1] Berl. Klin. Wochenschrift, 1876, No. 49.

[2] British Med. Journal, 1874, Nov. 21.

[3] Berl. Klin. Wochenschrift, 1877, p. 19.

[4] Clin. Soc. Trans. 1877, p. 57.

Suppuration appears to exercise a remarkable influence on the number of white corpuscles. This was first noted by Griesinger. During the process of suppuration, probably when a collection of pus is retained in the body, a remarkable number of leucocytes has been observed in the blood, disappearing when the pus escapes.[1] A case lately occurred in University College Hospital,[2] in which the blood of a patient recovering from perityphlitis, contained as many as 150 white corpuscles in a not over-crowded field: a few days afterwards a large abscess pointed in the back. The corpuscles, after the opening of the abscess, rapidly diminished in number and soon reached the normal. There was no enlargement of spleen or lymphatic glands. Apolant,[3] has observed, in a child four months old, with a large number of suppurating abscesses, that the white corpuscles were to the red as one to twenty, and that after the suppuration had for the most part ceased, they were as one to three hundred. A slighter excess, twenty to thirty in the quarter-inch field, may often be seen under similar circumstances, and I have observed in some cases that the majority of these corpuscles present fatty degeneration. This suggests that probably the excess may be due to the return of the leucocytes (or pus cells) into the vessels. A similar effect has been observed to follow the application of a blister. Septicæmia may also be accompanied by a considerable increase of leucocytes in the blood.

Local growths, composed of small cells resembling, in size and appearance, white blood-corpuscles, may be accompanied by a considerable excess of cells in the blood identical in appearance with the colourless corpuscles. In a case lately under my own observation of a large ulcerating lymphadenoma of the cervical glands without evidence of general gland disease, fifty to seventy colourless corpuscles were present in every field of the microscope. The case of Lücke's has been already referred to, in which, with a lymphadenoma of the axillary glands, the number of colourless and coloured corpuscles in the blood was equal. In cancer there may be a remarkable excess, as in a case recorded long ago by Dr. Fuller.[1] I have seen as many as seventy white corpuscles in a one-fifth inch field, in a case of cancer of the liver.

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[1] Several observations of this character are published by Bonne. Variation du nombre des globules blancs du sang dans quelques maladies. Paris, 1876.

[2] Lancet, 1876, ii. p. 253.

[3] Virchow's Archiv. Bd. lix. p. 302.

Pregnancy, &c..—A slight excess of leucocytes is common in the blood of pregnant women; the increase is, however, rarely considerable. The proportion of white to red has been carefully estimated for me in a series of cases by Dr. R. S. Miller. Seven were multiparæ and four primiparæ. The proportions were as follow:—

MULTIPARÆ.

Proportion of white to red.	Age.	Month of Pregnancy.
1—404	20	9th
1—400	28	8th
1—366	31	8th
1—360	37	7th
1—320	32	?
1—290	22	6th
1—213	27	6th

PRIMIPARÆ.

1—261	24	6th
1—200	20	8th
1—180	18	9th
1—130	17	7th

These results suggest that the increase is greater in primiparæ, and is greater the younger the individual.

A more considerable increase has been noted by Malassez[2] a few hours after delivery. Certain puerperal diseases may be attended with a great excess of white corpuscles, as was pointed out in 1844 by Bouchut, who regarded them as pus cells. Malassez observed an increase in fatal cases of puerperal septicæmia, amounting once to one white to fifty-four red. Schulten has recorded cases[3] in which, during an irregular febrile attack which commenced three days after delivery, there was a large excess of white corpuscles of various sizes, the proportion rising to one white to ten red. The normal state was regained when the fever subsided.

Many acute diseases are attended with the presence of an excess of pale corpuscles in the blood, an excess which is both relative and absolute. The excess does not bear any relation to the pyrexia, but seems related rather to the tissue changes which occur in the disease; and it is especially marked in those diseases which are accompanied with suppuration or with acute glandular swellings.

In typhoid fever, a marked excess has been noted by many observers.[4] Bonne states that it precedes ulceration, and disappears when this has been established. The maximum observed by him was 62,000 per cubic millimeter, an increase to four times the normal quantity. In relapsing fever an excess was observed by Allen Thomson[1] and others, and by Wilks in typhus fever. In erysipelas and pneumonia, a marked excess has also been observed.

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[1] Path. Trans. vol. ii. p. 224.

[2] Bull. de la Soc. Anatom. 1873.

[3] Virchow's Archiv. Bd. xiv. p. 505.

[4] Virchow, Bourdon, Jaccoud, Golgi, Bonne.

In glanders, a great increase is commonly present. This was pointed out by Delafond twenty years ago, and more recently by Christot and Kiener,[2] who found the increase occasionally amounted to the proportion of one white to six red.

In scarlet fever, a considerable increase was noted about the seventh day by Bonne, amounting in one case to 210,000 per cubic millimeter (about twelve times the normal). In diphtheria, also, an excess was found in a large number of cases by Bouchut. In variola, a marked increase was found by Duroziez and Brouardel, and it has been shown that this increase precedes the formation of pus in the eruption. Lambert found that in one case, on the third day of the eruption, the white corpuscles amounted to 10,000 per cubic millimeter, and that on the fourth day it had risen to 93,000, and on the fifth had fallen again to 30,000.[3]

In tuberculosis, a slight excess was found by Duroziez; in several cases fifteen, and in one forty, white corpuscles were seen in the field of the microscope. The occurrence of an excess of pale corpuscles in the blood in inflammatory affections of bone has been already mentioned.

Leucocythæmia in Animals.—In the domestic animals leucocythæmia may present precisely the same symptoms as in man. The cases have been carefully studied by Prof. Böllinger,[4] of Zurich, who has recorded several cases in the dog; and other cases have been met with in the pig, the cat, the horse, and the guinea-pig. The spleen has been in some cases affected alone; in others, the lymphatic glands have also been involved. The marrow of bones has also been observed to be changed. Most cases present secondary changes in the liver, kidney, or lung, and the same tendency to hæmorrhage has been present as is seen in man. Inoculation experiments failed.

DIAGNOSIS.—The recognition of developed splenic leucocythæmia is a matter of no difficulty. The general appearance of the patient is that of one suffering from a cachectic disorder; the abdominal trouble is sufficiently pronounced to attract attention; a drop of blood drawn from the finger is strikingly pale, and placed under the microscope is seen to contain so many white corpuscles that, at first sight, it may appear to consist chiefly of them. But in the early stage the disease is much less easy to recognise, and its diagnosis must, in some cases, be allowed for a time to remain in doubt.

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[1] Cormack, Nat. Hist. of the Epidemic Fever. Lond. 1843, p. 113. They were regarded as pus cells.

[2] Compte Rendue de l'Acad. des Sciences for Nov. 23, 1868.

[3] Quoted by Bonne. Lymphoid growths closely resembling those of leucocythæmia were found in a case of variola by Wagner in liver and spleen.

[4] Virchow's Archiv. Bd. lix. Heft 3 and 4. 1874.

The line of demarcation proposed by Magnus Huss, that the proportion of one in twenty is necessary to render a case one of leucocythæmia, is of limited application. It is only when the excess reaches this degree that the nature of the case can be pronounced with certainty. But in the early stage of every case there is a period in which the proportion is less than this. It is precisely in these early stages that the recognition of the disease is of the greatest practical importance, for it is then only that permanent good can be expected from treatment. The points on which the diagnosis must turn in these cases of commencing leucocythæmia are the recognition of a progressive increase in the white corpuscles of the blood, associated with a distinct enlargement of the spleen. The determination of the degree of blood change is, therefore, a matter of great importance.

The fact of an excess of pale corpuscles, and its degree, are usually determined by observing the number of such corpuscles in the field of the microscope. This is a rough method of very inexact character, and liable to many fallacies. The significance of the number will depend on the amount of blood in the field, *i.e.*, on the size of the field and the depth of the film of blood observed. The size of the field varies with every microscope and with every lens. The thickness of the film varies with the size of the drop and the weight of the covering glass. The only method of ascertaining the degree with accuracy is to count the numbers of red and white. This may be done absolutely, in a manner to be described directly, or relatively, by counting the numbers in a field or a series of fields. From the manner in which the corpuscles run together into rouleaux and masses, it is extremely difficult to count them in the blood as drawn. In order to do this accurately, it is necessary to prevent the red globules running together by diluting the blood with water, or better, with a one per cent. solution of common salt. A small quantity of solution, two or three times the volume of the blood, is sufficient. The separated red and white globules can then be counted. The operation may be facilitated by the employment of a stage micrometer, or a "quadrilled ocular," *i.e.*, an eye-piece micrometer ruled in squares; so that the field is divided into smaller areas. In order to obtain an accurate result at least as many corpuscles should be counted as will give one hundred white ones.

This relative proportion of white and red corpuscles is of great practical importance, since the two are, as has been seen, mutually related. But it is also important to know the actual alteration this represents, *i.e.*, what is the absolute number of white and red corpuscles in a given volume of blood. The determination of this point is of importance in prognosis as well as in diagnosis. The diminution of red corpuscles in leucocythæmia may reach a point equalled in no other disease, and on this diminution many of the consequences depend. It is evident, therefore, that to ascertain the degree and the course of the anæmia is of as much importance as to ascertain the degree and course of the leucocythæmia. The relative enumeration gives also little information concerning the absolute increase of white corpuscles. If, for instance, the corpuscles are found to be one white to fifty red, it cannot be assumed that the white are increased to six times the normal, because it may be, as is not unfrequently the case, that the red are reduced to one-third of the normal number and the increase in the white is only double.

It is only within the last few years that we have had at our disposal instruments which permit us to make these observations. The labours of Potain, Malassez, and Hayem in France, succeeding those of Vierordt, Welcker, and others in Germany, have made the method of enumeration of corpuscles available for clinical purposes.

The method formerly employed, first by Donné,[1] and subsequently by Vogel,[2] Robertson,[3] and others, consisted of allowing the slow subsidence of the corpuscles in defibrinated blood, placed in a narrow vessel, and observing the relative thickness of the layers of red and white corpuscles. But this method gives very inexact results, because the red corpuscles fall into closer contact than do the white, and hence the relative increase of the white is exaggerated. Another method is that which was devised by Welcker, and consists in employing the colour of the blood as a test of the number of red globules; the tint of a definite dilution of the blood, dried on white paper, is compared with that of a colour-scale, and the degree representing the amount of colouring matter is taken as a guide to the number of corpuscles. But this affords information only as to the amount of hæmoglobin contained in the blood—a very inexact guide to the number of corpuscles. The corpuscles are sometimes unusually pale, and the blood thus contains far less hæmoglobin than should correspond with the number of globules it contains. On the other hand, the corpuscles are sometimes smaller than natural, and thus, although not unduly pale, the amount of hæmoglobin is far less than should correspond to the same number of corpuscles. As an illustration of this, I may mention a case of anæmia in which I found the amount of hæmoglobin to be only thirty per cent. of the normal, while the red corpuscles were seventy-five per cent. of the normal average. They were rather small (on measurement) and pale.

The method of ascertaining the absolute number of corpuscles, white and red, in the blood is essentially that which was devised a quarter of a century ago by Vierordt, and consists in making a definite dilution of a measured quantity of the blood, and counting the number of corpuscles contained in a certain volume of that dilution. Several modifications of the process have been made, relating especially to the latter part of the process. Vierordt drew uniform lines of the diluted blood along a glass slide, and, after drying, counted the corpuscles in a given extent of those lines. Cramer substituted for this what may be termed a capillary cell, and in the better known methods of Potain and Malassez a capillary tube is employed.

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[1] Loc. cit. 1844.

[2] Virchow's Archiv. t. iii. 570.

[3] Bennett, loc. cit.

Hayem and Nachet have improved upon this by employing a cell of a certain depth. In the writer's modification of Hayem's instrument,[1] the area of the dilution in which the corpuscles are counted is determined by divisions on the glass slide at the bottom of the cell, and these divisions are of such a size, and the dilution is in such a degree, that each square corresponds to the ·00001 of a cubic millimeter of blood, and the average number of corpuscles in ten squares multiplied by 10,000 is the number per cubic millimeter of blood. The average of healthy blood was found by Vierordt and Welcker to be 5,000,000, and later results agree with this sufficiently nearly to justify the adoption of this number as the standard. This is the common mode of stating the corpuscular richness of the blood. By this method of counting, however, a much simpler mode of statement is obtained. The average number of corpuscles in two squares (·00002 cubic millimeter of blood) is in normal blood 100. I propose to take this quantity as the "hæmic unit," and the average number of corpuscles per hæmic unit is the percentage proportion to the normal.

The number of white corpuscles varies under normal circumstances from 5,000 to 15,000 per cubic millimeter of blood, *i.e.*, ·1 to ·3 per hæmic unit.[2] For the convenient record of the variations in the number of corpuscles in these cases I have arranged a chart, similar to a temperature chart, of which the two lowest divisions are subdivided into tenths for the record of the number of white corpuscles when they are only slightly in excess of the normal. A broken line at 100 is the average number of corpuscles in health; a dotted line at ·3 indicates the normal maximum of white corpuscles.[3]

Considerable caution is necessary in estimating the degree of leucocythæmia from the post-mortem appearance of the blood. The relative bulk of the red and pale portions of the clot constitutes a most fallacious guide, since the bulk of the latter depends mainly on the quantity of fibrin which it contains. Even microscopical examination of portions of the clot is not free from fallacy, since the pale corpuscles aggregate together in such quantities that portions of the clot appear to consist entirely of them, when their excess is moderate. A curious illustration of the difficulty of estimating the excess of pale corpuscles, post-mortem, was afforded by a debate at a French society, in which very different opinions were expressed by pathological authorities regarding the degree of leucocythæmia which was indicated by certain clots. In another case, in which the white and red corpuscles were supposed, from post-mortem appearances, to be in equal numbers, their proportion, just before death, had been found, on actual counting, to be as one to six. The only satisfactory way of examining the blood post-mortem is to take a portion from a vessel in which coagulation has not occurred.

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[1] This modified "hæmacytometer" consists of—(1) A small pipette, holding exactly 995 cubic millimeters. (2) A fine capillary tube, holding five cubic millimeters. (3) A small glass jar, in which the dilution is made. (4) The cell, above-mentioned, exactly one-fifth of a millimeter deep, the floor of which is ruled in tenth of a millimeter squares. Various diluting solutions have been recommended in order to change as little as possible the aspect of the corpuscles. It is not well, however, to endeavour to observe the characters of the corpuscles during the numeration. Whatever solution is employed the corpuscles are, more or less, changed by it. One which answers very well is a solution of sulphate of soda of a specific gravity of 1025.

The mode of proceeding is simple. A pipetteful of the solution is placed in the mixing vessel. Five cubic millimeters of blood are drawn into the capillary tube from a drop in the finger, and then blown out into the solution. The two are well mixed by a glass rod; a drop of the dilution is placed in the centre of the cell, the covering-glass applied and secured by springs, and the slide placed on the stage of the microscope. The lens is then focused to the squares. In a few minutes the corpuscles have sunk on to the squares. The number in ten squares is then counted.—Vide Lancet, Dec. 1, 1877.

[2] Their number, when not numerous, may be best ascertained with the hæmacytometer by observing the number of squares in each field of the microscope, and counting the number of white corpuscles in ten or twenty fields. By this means the average number per cubic millimeter or hæmic unit may easily be ascertained. The white corpuscles may be readily recognised by raising the objective until the corpuscles are fading out of focus, when the greater refractive power of the white corpuscles will render them conspicuous objects.

[3] The hæmacytometer and charts are sold by Hawksley, 300, Oxford Street.

Differential Diagnosis.—Leucocythæmia has to be distinguished (*a*) from diseases which resemble it in general characters, but differ from it in the absence of any excess of pale corpuscles in the blood, and also (*b*) from diseases, the general characters of which differ from those of leucocythæmia, but in which there is such an excess of corpuscles in the blood.

(*a*) Of the former class simple splenic anæmia, anæmic splenosis, as it might be termed, is that which is most likely to be mistaken for splenic leucocythæmia. In splenic anæmia there are the same tumour, the same anæmia, and many of the same constitutional symptoms, œdema, and even hæmorrhage. The distinction between the two diseases turns on the presence of a considerable or increasing excess of pale corpuscles in the blood. It has been repeatedly pointed out in this article that the distinction is, as far as we are able to observe, one of clinical character, rather than of pathological origin. The leucocythæmic form may be paralleled, most closely, even in its visceral changes, by an anæmic form of splenic change. Moreover, cases are seen in which every degree of leucocytal excess is present, and many of these are on the boundary line between the two affections, some stationary there, others progressing from the anæmic to the leucocythæmic form.

Spleno-lymphatic leucocythæmia may be mistaken for Hodgkin's disease. In most cases of the primary splenic form the glands are affected late in the disease, and in slight degree, and the spleen is usually very large and regular. In Hodgkin's disease the affection of the glands is the constant occurrence, and the spleen is rarely very large, and is sometimes, though not often, irregular in form. The great excess of pale corpuscles common in splenic leucocythæmia is rarely met with in Hodgkin's disease. In the latter the proportion of one to four is seldom reached, and very rarely, if ever, exceeded; in the former it is frequently exceeded. In Hodgkin's disease the pale corpuscles are, as a rule, smaller than in the splenic affection.

The cases of lymphadeno-splenic leucocythæmia, in which the enlargement of the glands is an initial symptom, occurring with the spleen, the splenic enlargement being great and the excess of leucocytes very considerable, often present much difficulty in their diagnosis. They are really intermediate forms between splenic leucocythæmia and lymphadenosis, the result of a general affection of lymphatic tissues. The larger and smoother the spleen, the earlier it enlarges, the more rapid and extensive the leucocythæmia, the more closely does the disease approach the characters of primary splenic leucocythæmia. The slighter the enlargement of the spleen, the later it occurs, and the slighter the blood change, the more closely does the case approach the variety of lymphatic affection distinguished as Hodgkin's disease.

(*b*) It is seldom that splenic leucocythæmia has to be distinguished from diseases altogether different in general character. The rare cases of leucocythæmia without splenic change can only be distinguished from simple anæmia by a microscopical examination of the blood. In tuberculosis, the pale corpuscles may be increased in number even to three times the normal, but the increase is not progressive and enlargement of the spleen is rare and never considerable.

In some forms of cancer there is a considerable excess of pale corpuscles in the blood. If the cancer be of the abdominal glands or near the spleen, some doubt might exist as to the existence of a splenic leucocythæmia. A careful examination of the tumour would, however, in most cases decide the question.

In suppuration it must be remembered that there may be a large excess of pale corpuscles in the blood. As many as a hundred and fifty have been observed in a field. (See p. 287). The excess is transient, and disappears soon after the abscess is opened.

In peculiar states of the system, a hæmorrhage may cause a considerable excess of white corpuscles to be present in the blood, and the condition might be thought to be one of primary leucocytal excess. The extent of the hæmorrhage, the transient character of the blood-change, and the absence of splenic and glandular enlargement, will prevent an error.

PROGNOSIS.—Leucocythæmia is to be regarded as the indication of a disease of the blood-forming organs, depending on an alteration in their structure, which tends to increase, and tends also to extend to other organs, the functions of which are more or less affected thereby. The course of the disease is thus progressive, until a degree of change is reached incompatible with life. At present no means of arresting this progress has been discovered, and the ultimate termination of the developed disease is only too certain.

The prognosis is, however, less immediately grave, as there exists a disproportion between the amount of blood-change and the extent of the organic disease with which it is associated. The prognosis is best in those rare cases in which there is an excess of white corpuscles without any disease of spleen, glands, or bones with which it can be associated. An example of this is the case recorded by Dr. Lloyd Roberts, and before alluded to.

The prognosis is less grave in the early stages of the leucocythæmia. When the blood-change and organic disease are both considerable, the downward progress of the case sometimes varies in its rate, and may be retarded, but it is usually uninterrupted. There are, however, many cases on record in which a condition identical, in all recognisable features, with that of commencing leucocythæmia, has lessened and even passed away under treatment. They are cases in which the splenic enlargement is moderate, the blood-change slight, and in which there are no signs of general cachexia, no profound anæmia, no tendency to œdema or hæmorrhage.

In cases of pronounced splenic leucocythæmia, in which the disease has led to general symptoms, considerable blood-change, œdema or hæmorrhage, the prognosis is of the gravest character, and death may be anticipated sooner or later as the certain termination. But it is important to inquire what indications of rapidity of course and proximate danger are afforded by the several symptoms.

Etiological conditions afford little prognostic indication. Neither sex nor age influences materially the duration of the affection, except that the course of the disease is a little more acute in women than in men, and apparently more rapid when it commences between the ages of forty and fifty, than when it commences at any other period. It is doubtful whether any other causal condition has much relation to prognosis. Instances of slow and rapid course are recorded due to almost every cause. It has been said that cases which are due to malarial poisoning run a more chronic course than others. This is, however, very doubtful. If the period during which a splenic tumour existed, when there was no leucocythæmia, is excluded from consideration, the course of the malarial leucocythæmia has been, in many instances, a very rapid one.

Among the symptoms of leucocythæmia which afford prognostic indications, one of the most important is the state of the blood. The greater the excess of white corpuscles, the graver is the condition of the patient, the greater the liability to serious complications, to hæmorrhage, internal and external, and to the diseases of organs which are produced by the accumulations in them of the white corpuscles, and by the lymphoid growths. It may be doubted whether the asthenia, and the tendency to hæmorrhages, are not due as much to the deficiency of red corpuscles as to the excess of white, and it is probable that the numeration of the red corpuscles by the hæmacytometer, will afford more definite prognostic information than is forthcoming from a merely comparative observation. Other things being equal, the smaller the excess of white, and the larger the absolute number of red, the better is the prognosis.

The size of the spleen affords little information considered alone. If a large spleen is combined with a considerable blood-change, the case will probably soon terminate. Enlargement of the glands, if distinctly secondary to the splenic enlargement and blood state, is a grave symptom, indicating a widely-spread general change. If occurring simultaneously with the splenic affection, as in the cases of lymphadeno-splenic leucocythæmia, the course of the disease will probably be rapid, especially if the enlarged glands are soft. The cases in which the glandular enlargement is distinctly primary are considered in the next article.

The prognostic significance of hæmorrhage varies according to its seat and degree. When extensive, the loss of blood constitutes a source of immediate danger, and renders the prognosis worse by its influence on the anæmia. When moderate, its significance depends on its position. Epistaxis affords little indication. It is frequently an early symptom, and has occurred repeatedly during many years. Hæmorrhage from the bowels is of graver significance, and rarely occurs except when the general cachexia is developed, and then is not uncommonly the cause of death. Hæmatemesis is of even worse augury, and where it occurs commonly immediately precedes death. Cutaneous extravasations are also signs of a cachectic state, and occur only towards the end. Œdema is another grave symptom. It is rarely an early occurrence, and when it does occur at the commencement and is considerable, it indicates an early blood-change, and, usually, a rapid course. When it is slight, however, and shows no tendency to increase, it may exist for a long time, and affords little prognostic indication. Later on in the disease, it indicates, when considerable, a profound degree of anæmia and a speedy termination. It constitutes in itself a source of danger from its influence on the respiratory organs. Diarrhœa, if persistent, is of grave significance; it lessens the patient's strength, and is also not unfrequently the immediate cause of death.

TREATMENT.—Developed leucocythæmia, associated with distinct changes in the lymphatic organs of the body, has hitherto proved one of the most intractable of maladies. Its progressive course has been arrested by no method of treatment which has been employed. In some cases, however, the patient's condition has been improved, and the course of the disease apparently retarded, and there is reason to believe that, at a much earlier period than that at which the affection usually comes under notice, it has been permanently arrested, and even cured. Further, the little knowledge we possess of its causes suggests that something may be done to prevent its occurrence, and the equally slight knowledge which we have obtained of its pathology does not preclude the hope that means may yet be discovered of arresting its developed course.

The prophylactic treatment of leucocythæmia resolves itself very much into the prevention of its causes. The condition to which it is more distinctly related than to any other is certainly the occurrence of intermittent fever. It is more frequent in ague districts than in others, and it may apparently occur as an effect of malaria which has not produced intermittent fever. Its rarity in this country may not improbably be associated with the practical disappearance of intermittent fever.

There is reason also to believe that the process of the intermittent does, itself, set up changes which result in leucocythæmia. Many of the cases have succeeded long and repeated attacks of ague, extending, in several, over twenty years. From such attacks we know that a permanent splenic tumour results, as the consequence, apparently, of the repeated congestion of the organ. It is reasonable, therefore, to regard this splenic tumour as the connection between the intermittent and the leucocythæmia, and to believe that by abbreviating attacks of intermittent, and obtaining, by every available means, early contraction of the spleen, the chances of subsequent leucocythæmia may be lessened. The means for this purpose are, it need hardly be said, the free administration of quinine in large doses, care being taken to secure its absorption, and to this may be added the adoption of other measures to be immediately described for promoting the same end.

In all cases in which a chronic enlargement of the spleen exists, great care is necessary to avoid all causes of congestion of the organ. Of these, exposure to cold is one of the most powerful, and is especially hurtful. Cases are on record in which exposure to cold was the only apparent exciting cause of a splenic tumour, and others in which the same cause was the excitant of a fatal leucocythæmia. Besides the measures to be indicated for inducing contraction of the spleen, its congestion may be lessened by the avoidance of all causes of portal congestion, by strictly regulated diet, the prevention of constipation, and by the relief of portal congestion, if it should occur, by aperients.

The precautions indicated are especially needed in women who have been exposed to malaria, or possess any splenic enlargement, during their menstrual or parturient periods. The profound systemic disturbances at these periods have a distinct influence in the production and intensification of leucocythæmia. Such women should observe the greatest care at the menstrual period, to avoid cold, fatigue, mental disturbance, and constipation. If they bear children their strength should be carefully kept up during pregnancy, the loss of blood at parturition as much as possible prevented, and subsequent recovery hastened by hæmatinics. Lactation should be absolutely prohibited.

In some cases simple deficiency of red corpuscles has preceded the increase in the white. It is of the highest importance to remove this deficiency. It must be confessed that its removal is, when associated with splenic enlargement, often as difficult as is that of leucocythæmia, and it may be indicative of quite as profound change in the splenic tissue. An alteration in the spleen, by which pale corpuscles are no longer formed or cannot escape, is almost as grave as one in which they can, although in altered form, still pass into the blood. For this anæmia iron, abundant nitrogenous and easily assimilable food are the customary measures, but it is of the greatest importance that these should be conjoined with an attempt to obtain reduction in the size of the spleen. Alone, hæmatinics are commonly unsuccessful in the treatment of splenic anæmia. In observing their effect, the hæmacytometer is of great utility. The actual number of corpuscles is the best guide to the effect of remedies, and a distinct rise in their number may be evidence of improvement earlier than that afforded by the aspect of the patient or the tint of the mucous membranes. In one case I found iron entirely useless until contraction of the spleen was obtained by galvanism, and with the reduction in the size of the organ (to about two-thirds of its former size) there was a corresponding rise in the number of blood-corpuscles. When iron fails, phosphorus, and arsenic may be given.

The treatment which has been adopted for pronounced splenic leucocythæmia has been of five kinds. First, to promote splenic contraction; some agents for this purpose also have an action on the blood. Secondly, to improve the nutrition of the blood. Thirdly, the use of remedies which have a general influence on the nutrition, but have been given, in leucocythæmia, more or less empirically; of these the most important are iodide of potassium, phosphorus, and arsenic. Fourthly, the substitution of healthy for the diseased blood by transfusion. Lastly, the removal of the diseased spleen by operation.

(1) The reduction of the bulk of the spleen has always been one of the principal objects in the treatment of the leucocythæmia. It has been chiefly based on the notion that the spleen was the source of the excess of white corpuscles, that the degree of leucocythæmia was proportioned to the enlargement of the spleen, and that a reduction in the bulk of the organ would be attended by an improvement in the state of the blood. Undoubtedly there is an accumulation of lymphoid corpuscles in the spleen, and these probably grow and multiply there as they do not in the blood. The larger the spleen the greater the amount of corpuscles within it, and the larger the quantity of leucocytes retained in the splenic pulp, the further this is from its normal condition and function. Contraction of the spleen is attended, even in simple splenic tumours, with an expulsion of cells from it, and an increase of white cells in the blood. Thus there is reason to believe that the production of leucocytes in the spleen may be lessened by contraction, just as it is increased by dilatation and congestion. But when the spleen is greatly enlarged, when its tissue is altered by the lymphatic and connective tissue overgrowth which unquestionably occurs, it is difficult to see how any material change can be effected in the formation or transformation of the lymphoid cells by obtaining a slight contraction in the organ. In the earlier stage of the affection, however, when the lymphatic overgrowth in the organ is trifling, and the tissue elements but slightly altered, a considerable contraction of the spleen may expel a proportion of the contained leucocytes, and, by altering the relation of the rest of the tissue to

the blood, the transformation of the corpuscles may be aided. The fact just mentioned regarding splenic anæmia renders it probable that thus permanent improvement, even cure, may be effected, of a condition which may reasonably be regarded as one of commencing splenic leucocythæmia.

Experience is in accord with these conclusions. When leucocythæmia is developed and the spleen is large, no distinct good has been effected by such reduction in the size of the spleen as can be produced under the circumstances. In cases in which the affection has been less advanced, in which the blood-change is slight, and the splenic enlargement is inconsiderable, very remarkable improvement has resulted from this treatment, amounting in some cases to a disappearance of all the symptoms of the disease. It is of course open to some doubt whether these were actually cases of commencing leucocythæmia, but it seems clear that they cannot be distinguished from the early stage of cases in which characteristic symptoms subsequently developed.

Quinine must unquestionably be placed first among the internal remedies for this purpose. It possesses a double influence. It is capable (*a*) of acting directly on the spleen, and, apparently (*b*) of influencing the vital properties of the corpuscles. (*a*) The normal spleen of dogs[1] and pigs,[2] and the enlarged spleen of man, can be made to contract by its use. It has been suggested that its action is directly on the contractile tissue of the spleen, and this is probable from the fact that the contraction can be obtained after section of the splenic nerves.[3] Jerusalimsky has however asserted that after division of the nerves the contraction produced by quinine is slighter, although it occurs earlier, than when the nerves are untouched, and he suggests that the primary action of the quinine may be on the peripheral nerves and muscular tissue in the spleen, its second action being on the splanchnic and central nervous system. (*b*) The action of quinine on the white corpuscles is to lessen their amœboid movements and arrest their migration through the walls of the blood-vessels. In the frog, Binz found that inflammation of the mesentery was attended with an excess of pale corpuscles in the blood and enlargement of the spleen, and that both lessened on the injection of quinine. He suggested that quinine might cause reduction of the size of the spleen in man by interfering with the production of the pale corpuscles, but there are no other facts to support the view, and the slight utility of quinine in developed leucocythæmia, as Mosler points out, is opposed to Binz's theory. But without going so far, it is certainly possible that a remedy which has a distinct effect on the vital properties of the corpuscles may influence their production and metamorphosis, and that its beneficial effect may be in part due to this influence. Jerusalimsky asserts that after the application of quinine only one nucleus could be rendered visible in corpuscles, in which, before, three were to be seen.

[1] Mosler, Binz, Jerusalimsky. [2] Kuchenmeister. [3] Mosler, Jerusalimsky

It is in the early stage of the disease that quinine has appeared to be of real service. Mosler records a case of enlarged spleen with an excess of pale corpuscles in the blood, their proportion to the red being one to twenty, in which, after several days' intoxication with quinine, the excess of corpuscles diminished and the spleen lessened in size, and the improvement in both went on to complete recovery. A case in which a cure was apparently effected in which the proportion of white to red was one to thirty or forty, has been recorded by Ehrlich. Farre[1] has recorded a case of moderate splenic tumour, with an "excess" of white corpuscles in the blood, in a woman who had suffered from a tertian ague for a week, a year before. On quinine and iron there was a rapid disappearance of both symptoms. Great improvement occurred also in a case described by Hewson,[2] in which the enlargement of the spleen was moderate although the excess of pale corpuscles was great. But in some cases in which the spleen was lessened in size by the quinine, the course of the disease was not thereby retarded. In other cases, again, quinine, in large doses, has been without influence even on the spleen.[3] It is important to give the quinine in large doses, producing and maintaining its full toxic effect. Mosler recommends the hydrochlorate, on account of its greater solubility. Its hypodermic injection does not seem to possess any special advantages.

Ergotine has been employed for the same purpose by Da Costa.[4] Great and permanent improvement was effected in one case, in which the enlargement of the spleen was slight, and the increase in white corpuscles trifling—not more certainly than accorded with the anæmia which existed. In another case, in which the leucocythæmia was decided, a distinct diminution in the size of the spleen was obtained, but the patient died soon afterwards, the course of the disease being apparently uninfluenced. The ergotine was administered by subcutaneous injection, the dose being 5–10 grains every second day.[5] The solution employed was of 40 grains of ergotine, 30 mimims of glycerine, and water to 220 minims.

Piperine has been found by Soenderop to produce contraction of the spleen of the dog, and his observation has been corroborated by Mosler. The latter has given piperine in leucocythæmia in doses of 8 to 30 grains, in combination with quinine and eucalyptus.

Mercurial inunctions over the spleen were employed by Addison,[6] and some improvement followed, but they were combined with the internal administration of quinine. Wilks found mercurial inunction useless.

The application of electricity to the spleen of an animal is a ready and certain mode of causing its contraction; and an enlarged spleen in man may also be lessened in bulk by its means.[7] It has been asserted by Botkin that extreme reduction in size may thus be obtained, but it is certainly only in very exceptional cases that this can be produced. Faradisation is the readiest form, and is an effective means of causing contraction of the exposed spleen of an animal, and this is the variety which has usually been employed in splenic tumours, but it has failed entirely in the hands of some observers.[1] I believe the interrupted voltaic current to be a much more effective agent. The passage to deeper parts of a strong faradaic current may be almost completely arrested by a thin superficial layer of muscle, which contracts first, while the voltaic current penetrates much more deeply, and is not arrested in a similar manner. In a case of enlarged spleen with simple anæmia, in which I made some careful observations, I found that each application was followed by an immediate reduction in the diameter of the spleen by about one inch, increasing for about half an hour, and remaining for several hours and then passing off. The subsequent increase afterwards did not quite reach the former size, and the habitual size of the organ gradually lessened, until in a fortnight it was permanently reduced about two inches in width. No other treatment was employed at the time, and until the commencement of the application the size of the organ had remained the same. In another case in which the spleen reached the level of the umbilicus and had been stationary for three months, a few weeks' daily application so reduced the size of the organ that it could be only just felt below the ribs. The positive pole should be placed behind, over the tenth rib, and the negative pole on the tumour in front, the current being interrupted by a commutator, or by moving the negative electrode over, or dabbing it on, the organ. As strong a current should be employed as the patient can conveniently bear. In some cases of leucocythæmia even voltaism has failed to produce diminution in the size of the spleen.[2]

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[1] Lancet, July 6, 1861.

[2] Amer. Journ. of Med. Science, vol. xxiv. 1852, p. 365.

[3] Vidal, Gaz. Hebd. 1856, p. 167; Wilks, Guy's Hosp. Rep. 1859, p. 112.

[4] American Journal of Med. Science, Jan. 1875.

[5] A somewhat large dose of an active preparation.

[6] Lancet, Jan. 7, 1860.

[7] First employed by R. Wagner, Jenaische Annalen, 1849.

The cold douche causes immediate contraction of the exposed spleen of an animal: the organ becomes smaller, darker, and wrinkled.[3] The same effect is produced in slighter degree by the application of the douche to the abdominal wall in the neighbourhood of the spleen. It has been found also that in man a cold douche to the splenic region produces very distinct diminution in size of the enlarged spleen. It was employed as an adjunct to the treatment of ague by Currie,[4] and more recently by Fleury. In leucocythæmia, cold douches were recommended by Niemeyer, and also, on the above experimental grounds, by Mosler, who advises the employment of water at a temperature of 55° or 60° F., and an application of from fifteen to sixty seconds. The local application of ice has a similar effect, and may also be used. These means will not, however, alone effect the whole reduction in bulk of which the spleen is capable. In a case of eucocythæmia recorded by Mosler, the length of the spleen was reduced from twenty-five to twenty centimeters in length by cold applications, and then a further reduction to eighteen centimeters was effected by quinine.

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[1] Elias. Deutsche Klinik, 1874, No. 5; Andrae, Deut. Zeit. f. prakt. med., 1874, p. 29; Chvostek, Wien. Med. Wochenschrift, 1877.

[2] Chovstek, loc. cit.

[3] Mosler, Virchow's Archiv. 1873, vol. lvii. p. 1.

[4] Medical Report on the Effects of Water, Cold and Warm: London, 1805.

(2) The marked effect of iron in simple anæmia, even when the corpuscles are few in number, and the white relatively increased, led to its use in leucocythæmia. It was hoped that, since in simple anæmia it must increase the production of cells capable of being transformed into red corpuscles, or perhaps aid their transformation, it might have a similar influence in anæmia. Any expectation of benefit has, in the developed splenic form, been invariably disappointed. Slight improvement in the aspect of the patient has occasionally been noted, but no diminution in the blood state, or decrease in the spleen. The same inutility of iron alone may be observed in simple anæmia associated with lymphatic overgrowth. I lately had under treatment two cases of anæmia in girls, one of simple anæmia, the other with a large spleen. In each there was profound anæmia, the percentage of corpuscles being in one twenty-six and in the other thirty-six per cent. of the normal. There was no marked excess of white corpuscles in the blood of either. Each was placed on the chloroxide of iron. In the patient with no splenic enlargement, but with only twenty-six per cent. of corpuscles, a rapid improvement occurred. Every enumeration showed an increase in the number of corpuscles, until a percentage of seventy-five, and ultimately, with change of air, of ninety-two, was reached. In the other patient (alluded to previously), the corpuscles remained almost the same, the numbers obtained ranging between thirty and thirty-five per cent., not varying more than three or four per cent. until the spleen was reduced in size by galvanism, when they rose to forty-six per cent.

We know too little of the process of blood-making to infer from these facts that iron must always be useless in leucocythæmia. It is not therefore surprising to find that its influence in this disease should be similar to its effect in anæmia; that simple leucocythæmia, without lymphatic overgrowth, should be benefited by its use, just as is simple anæmia. In Dr. Lloyd Roberts' case a condition of extreme leucocythæmia unattended by any discoverable splenic enlargement or lymphatic disease, was cured by the administration of iron.

Vogel's experience induced him to believe that iron is of service in cases of commencing leucocythæmia in which the splenic enlargement is small. In its use more than usual care must be taken to prevent a constipating effect, since thus splenic enlargement may readily be increased.

Cod-liver oil has sometimes appeared to do good, especially when given in conjunction with iron. The combination of influences brought to bear upon patients at the mineral baths of Germany and elsewhere has seemed, in some cases, to be beneficial. Valentiner[1] mentions one case in which, at Pyrmont, an excess of white corpuscles, associated with a splenic tumour, disappeared. In other cases, however, similar treatment has been useless.

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[1] Deutsche Klinik, 1860, p. 200.

(3) Arsenic, from its general tonic influence and its power of influencing many obscure morbid processes, has been used in leucocythæmia, but without any distinct benefit.[1]

Phosphorus has been given in leucocythæmia on empirical grounds. It was first recommended in splenic tumour by Dr. Broadbent,[2] who had found that it was capable of producing some of the therapeutical effects of its congener arsenic. A case of enlarged spleen with anæmia and slight excess of white corpuscles presented, under its use, marked diminution in the anæmia and in the size of the spleen. In a case recorded by Dr. Wilson Fox a marked diminution in the excess of corpuscles was observed, and the patient's health appeared much improved. The patient, however, subsequently died from the affection. In a case of lymphadeno-splenic leucocythæmia I observed a reduction of the corpuscles from one white to four red to one white to one hundred red under its use; but there was reason to ascribe its effect to its influence on the lymphatic glands rather than on the spleen, and the patient died from renal disease.

This case suggests that it may exercise an influence by its known property of causing fatty degeneration; it may thus cause the early degeneration of the white corpuscles, or possibly of the growing cells from which they are formed, and thus may lessen their number. Those organic disturbances which depend upon the excess of leucocytes may thus possibly be lessened. But on the other hand some facts suggest that the formation of red corpuscles may, by its influence, be promoted. I have twice found that in lymphadenoma the percentage of red corpuscles steadily increased under its use. It is only at an early stage of the disease that such an influence can be looked for. In every other case in which the excess of white corpuscles was considerable phosphorus has not appeared to have any beneficial effect.[3] It is very desirable that in cases in which it is employed the numbers of red and white corpuscles should be observed and their changes followed by the hæmacytometer.

Iodide of potassium has also been given and found practically useless. Under its influence Mursick observed a transient diminution in the spleen and glands, but no permanent effect. Bromide of potassium, and mercury, the latter pushed to salivation, have also been apparently entirely without effect.

The fact that the pale corpuscles of the blood are increased during suppuration and diminish with the evacuation of the pus, led Apolant[4] to suggest that their number in leucocythæmia might be lessened by the formation of a freely suppurating surface. He made some observations on animals, from which he concluded that the number of pale corpuscles could be lessened by suppuration. The suggestion has not yet, I believe, been applied to the treatment of leucocythæmia, and there is no reason to expect that the gravest element in the blood state, the paucity of red globules, would be lessened by it.

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[1] Oppolzer, Bourdon, Valentiner, Berl. Kl. Wochen. 1865, 321.

[2] Practitioner, Jan. 1875.

[3] Sir W. Jenner, Clin. Soc. Discussion, Lancet, 1876, ii. p. 787. Moxon, Clin. Soc. Trans. 1875.

[4] Virchow's Archiv. Bd. 59, p. 302.

(4) The morbid state of the blood being, in most cases of leucocythæmia, the direct or indirect cause of death, it has seemed a reasonable measure to substitute healthy for the diseased blood by transfusion. The results of the operation have not, however, been very favourable. Transient improvement has followed; in a case of Mosler's there was a diminution in the size of the spleen and an improvement in the blood, but no lasting influence on the disease. In most cases the quantity injected has been small—four or six ounces—and it is evident that if the leucocythæmic state were well developed, as in all the cases in which it has been employed, the injection of a quantity which bears so small a proportion to the total mass of blood, can effect very little alteration. Repeated transfusion has therefore been suggested, but it has not yet been tried. The injection has been made in most cases into a vein, and the veins of the foot have been recommended as farthest from the heart. The farther from the heart a transfusion is made, the larger the quantity of air necessary to produce dangerous consequences. Arterial transfusion was recommended by Heute and tried at Breslau; defibrinated arterial blood was injected into the radial artery, with the result of causing sudden death, on which the post-mortem examination threw no light.[1]

(5) Excision of the spleen has been performed in a few cases of leucocythæmia in the hope that the disease might be thereby arrested.[2] In no case, however, has the patient survived even the immediate effects of the operation. Death has, in most, been due to hæmorrhage from vessels which were in a position (as in splenic adhesions) in which they could not be secured. In a few cases of simple splenic hypertrophy, without leucocythæmia, the operation has been successful. In leucocythæmia the tendency to hæmorrhage is unquestionably greater than in most cases of simple splenic anæmia; and this constitutes a danger so great as to render the operation scarcely justifiable in the advanced stages of the disease. Naturally, a risk of immediate death so great as is, and must be, involved in the operation, is only consented to when the disease is advanced, and the patient's danger one of which he is conscious by symptoms, such as œdema and dyspnœa, in themselves indicative of the greatest source of risk—the profound blood-change. We are yet, moreover, without any evidence as to whether the removal of the spleen would be attended with an improvement in the deficiency of red globules,

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[1] Schmidt's Jahrbuch, vol. clx., 1873, p. 50.

[2] Bryant, Guy's Hosp. Rep. 1866, p. 444; Ibid. 1867, p. 411; Kœberlé, Gaz. Hebd. 1867, No. 43; Browne, Lancet, ii. 1877; Billroth, Wien. Med. Wochenschrift. 1877. Other unsuccessful cases, in which leucocythæmia was not observed, or was noted to be absent, have been recorded by Quittenbaum, Kuchler, and Spencer Wells, and two successful cases by Péan.

which constitutes the gravest source of danger. It is much to be desired that in any future cases of splenotomy for simple hypertrophy, the changes in the blood state after the operation should be carefully followed by numeration of the corpuscles.

The several symptoms of leucocythæmia are often so urgent as to need special treatment. One is the frequent and severe hæmorrhage. This has to be checked by local pressure, cold applications, and astringents applied locally. The application of crystals of perchloride of iron, as recommended by Sir William Jenner for the hæmorrhage in hæmophilia, is one of the most powerful styptics. With these may be combined the hypodermic injection of two or three grains of ergotin. Hæmorrhage from the stomach and bowels must be treated in the usual manner; injection of ergotin, with astringents by the mouth. Vomiting is best relieved by a posture which lessens the pressure on the stomach, by the ordinary gastric sedatives, and especially by counter-irritation. The diarrhœa often needs strong and repeated astringents, tannin preparations, or lead, with opium. Constipation should be avoided, but gentle aperients only must be employed to counteract it, on account of the liability to, and danger of, diarrhœa. Splenic pain is best relieved by counter-irritation, sedative liniments, and hypodermic injection of morphia. It may often be lessened by the avoidance of the causes of gastric distension. For the œdema, diuretics and cardiac tonics, especially digitalis, are the most useful. Purgation, for its removal, is inadmissible. For the dyspnœa, inhalations of oxygen have been employed by Jaccoud and Sizer, and have been found to give relief, but only of a very transient character.

In all means employed, the utmost care must be taken to avoid any measures likely to lessen the patient's strength. Some of the symptoms are often misapprehended, and unnecessary and very injurious attempts are made to remove them. This is especially the case with the œdema, the abdominal fulness, and the "icterus lienalis." For all these free purgation is sometimes employed, and the patient's downward course is thereby rapidly accelerated.

HODGKIN'S DISEASE.

By W. R. Gowers, M.D.

Synonyms.—*Growth.*—Vascular Sarcoma of the Lymphatic Glands, Craigie; Malignant Lymphoma, Billroth; Lympho-Sarcoma, Virchow; Malignant Lympho-Sarcoma, Langhans; Lymphadenoma, Wunderlich, Ranvier; Desmoid Carcinoma, Schultz.

Disease.—Anæmia Lymphatica, Wilks (1862), (Hodgkin's Disease, Wilks, 1865); Pseudo-Leukæmia, Cohnheim, Wunderlich; Adénie, Trousseau; Lymphadénie, Ranvier; Lymphatic Cachexia, Mursick.

Definition.—Hodgkin's disease, or lymphadenosis, as it may be conveniently termed, is an affection characterised by a widely-spread enlargement of the lymphatic glands, commonly also by an enlargement of the spleen, and by progressive anæmia. The enlargement of the glands and spleen is due to the growth in them of adenoid tissue, having a tendency to undergo a fibroid transformation, and in the spleen to be localised in isolated masses. In many cases disseminated growths of similar structure occur in other organs, especially in the intestinal canal, the liver, and the kidneys. The blood presents invariably a diminution in the number of red blood corpuscles, occasionally an increase in the white corpuscles, usually inconsiderable.

History.—Malpighi[1] was the first writer to mention the association of general enlargement of the lymphatic glands with nodules in the spleen, but special attention was first called to the association as the salient feature of certain cases of disease by Dr. Hodgkin[2] in 1832. Of the series of cases Dr. Hodgkin published, some are clearly instances of other morbid states, but at least two are examples of the special disease now called after him.

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[1] De Viscerum, Lond. 1669, De Liene, cap. v. "De quibusdam corporis per Lienem dispersis." Speaking, p. 131, of the manner in which these bodies could be rendered visible, he says, "In homini difficilius emergunt, si tamen ex morbo universum glandularum genus turgeat, manifestiores redduntur, auctâ ipsarum magnitudine, ut in defunctâ puellâ observavi, in quâ lien globulis conspicuis racematim dispersis totus scatebat."

[2] Medico-Chirurgical Transactions, vol. xvii. 1832, p. 69.

Many previous writers had alluded to or described instances of general enlargement of the lymphatic glands, but most of these were regarded as scrofulous or cancerous. A well-described and remarkable example of the former was described by Morgagni, by whom it was related, in 1752.[1]

The condition appears to have been that characterised by Craigie in 1828, as "enlargement with induration" or "vascular sarcoma" of the glands; and he carefully distinguished between this enlargement with induration, non-caseating, and the caseating strumous enlargement which he afterwards designated "tyroma glandularum." He also drew an accurate distinction between it and cancer, with which it had, he says, been confounded.[2]

Dr. Bright noticed the frequency with which tubercular disease of the lymphatic glands was associated with that of the spleen, and alluded to Dr. Hodgkin's observations, detailing again some of Dr. Hodgkin's cases.[3] Velpeau, in 1839,[4] drew attention to the fact of the occurrence of glandular hypertrophy, independently of scrofulous disease.

The discovery of leucocythæmia, and of the fact that the lymphatic system participates in the morbid process, drew closer attention to the affections of the lymphatic glands. In 1848 Virchow published a case of general enlargement of the glands, without affection of the spleen, in which the clots, found post-mortem, appeared to contain an enormous excess of white corpuscles. Dr. Hughes Bennett, in reproducing this observation in 1852, drew attention to Dr. Hodgkin's earlier paper. Larrey in 1852 alluded to the disease in general terms, but adduced as an example the case recorded by Morgagni. A case was brought by Dr. Markham[5] before the Pathological Society in 1853, in which the first microscopical examination of the growths in the spleen was made by Dr. Bristowe. The condition, however, attracted little notice until a fresh series of cases was described in 1856 by Dr. Wilks,[6] who pointed out their resemblance to those described by Dr. Hodgkin.[1] In the same year an observation was communicated to a French Society by Bonfils, and in 1858 two characteristic cases were carefully described by Wunderlich,[2] who called attention to their distinction from tubercular and carcinomatous affections. The structure of the enlarged glands was carefully described by Billroth[3] in 1858. In 1859 a case was published by Pavy, and in 1860 Potain published a thesis on the effects of hypertrophy of visceral glands. In 1861, cases were published in this country by Mr. Jonathan Hutchinson, and abroad by Cossy,[4] Potain, and Perrin. Since then a large number of cases have been placed on record. In 1864 the pathological features and relations of the disease were briefly described by Virchow.[5] In 1865 Dr. Wilks[6] collected the cases which he had published, and compared their features; and in the same year another collection of cases was made by Cornil, and Trousseau[7] published a clinical lecture, in which he brought forward several new cases, and gave a general description of the disease. Wunderlich published a fresh collection of cases in 1866. In 1867 the microscopical characters of the disease were described by Ollivier and Ranvier, and by Billroth; and in 1869 fresh observation of its structure, by Dr. Burdon Sanderson, were included in an account of the history and symptoms of the disease by Dr. Murchison.[8] Since that time several small collections of cases have been published, in this country especially by Dr. Southey,[9] and in France, in the theses of Rousseau (1874) and Grocler (1873), and in Germany by Schultz and Langhans.

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[1] Epist. lxviii.; Cooper's Trans. p. 604. The case was that of a boy, aged 15, who observed the enlargement of the glands of the neck three months after a severe mental shock. The glands beneath the jaw, in the neck and thorax, were all enlarged, and tumours of the same character existed in the integuments of the abdomen, and in the abdominal cavity. The progress of the disease was rapid, and accompanied with fever. After death the tumours were found full of whitish matter, in some rather fluid, in others solid, and some were suppurating. The whole omentum was full of strumous tubera, and masses, from a hen's egg to that of a pigeon's in size, occupied the peritoneum, pancreas, and mesentery. There was also apparently similar growth in the tissue about each kidney. The glands in the small intestines were enlarged. One tumour, as large as a hen's egg, existed in the mediastinum and compressed the trachea. There were hard and stony globules the size of grains of barley on the surface of the lungs, and strumous tumours were situated in the costal pleura.

[2] Path. Anatomy, 2nd edit. 1828, Diseases of Glands, p. 250. "The great hardness and the malignant tendency of this growth have procured for it from most authors the ominous names of scirrhus and cancer. Though correct enough for all practical purposes these epithets are not justified by the anatomical characters." He describes it as consisting of amorphous granular masses united by dense filamentous tissue.

[3] Guy's Hosp. Reports, vol. iii. 1838, p. 405. These have been quoted as separate cases.

[4] Leçons Orales de Clinique, t. iii.

[5] Path. Trans. vol. p. 177.

[6] Guy's Hosp. Reports, third series, vol. ii. 1856, p. 114.

VARIETIES.—A distinction has been drawn between the forms in which the glands are hard and those in which they are soft. The more acute the process the softer are the glands, the greater is the proportion of cells in them, and, it has been thought, the greater is the tendency for the cells to pass into the blood, and to persist there as an excess of white corpuscles. Hence the softer varieties have been regarded as examples of "lymphatic leucocythæmia," and distinguished from the harder forms, which have been termed "lymphosarcoma." But it will be seen that there may be no excess of leucocytes in the blood, even when the glands are soft; and there may be an excess when the glands are hard; that the same case may present both hard and soft glands, and that the two conditions may even be but different stages of the same affection.

Varieties may be distinguished according as the glandular affection exists alone or is associated with enlargement of the spleen, and as the latter depends upon the presence of limited growths, or of a diffused increase in the pulp tissue. The distribution of the glandular enlargement also constitutes a salient distinction between different cases. In some it is general, and uniform in degree; in others, certain glands are much more enlarged than others, and these may or may not be the glands first affected. In some cases, again, one group of glands may be the seat of great enlargement, and constitute a tumour having the characters of a local growth. These varieties of distribution may be thus classified—

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[1] Receuil des Travaux de la Société Médicale d'Observation, t. i. p. 157.

[2] Archiv für physiologische Heilkunde, 1858, vol. ii. new series, p. 123.

[3] Beiträge zur path. Histologie, 1858, p. 168.

[4] Cossy, Echo Méd. de Neuchâtel, 1861, t. v. Nos. 13 and 14, extracted in Gaz. Hebdom. Dec. 20, 1861.

[5] Krankhaften Geschwülste, vol. ii.

[6] Guy's Hosp. Reports, 1865, vol. xi. p. 56.

[7] Clinique Médicale, t. iii.

[8] Path. Trans. vol. xxi. 1870.

[9] St. Bartholomew's Hosp. Reports, vol. ix. 1873, p. 46.

1. Local growth only.	
2. Local enlargement preponderating.	*a.* primarily *b.* secondarily.
3. General uniform affection.	*a.* secondarily general. *b.* primarily general.

The first and last stand at the extreme limits; in the one the causes of the disease act solely on one part, in the other they act with equal force on all the lymphatic glands of the body.

Each of these varieties may be associated in any degree with an affection of the spleen, and with growths in the organs of the body, and each with an excess of white corpuscles in the blood. The first, however, in which the glandular growth is limited to one set of glands, appears to possess the characters of a local growth, and it is doubtful in what relation they stand to the cases in which the glandular disease is more widely spread. Those cases are, therefore, described in this article incidentally, and only those cases are analysed in which several groups of glands were diseased.

Growths in the viscera, such as occur in association with glandular disease, sometimes also occur alone, either limited to one organ or occurring in several. These are likewise discussed incidentally, and only such cases are included in the analysis as presented also enlargement of several groups of glands.

ETIOLOGY.—Recorded cases throw very little light on the causes of lymphadenosis. In a large number of cases the affection commenced while the patient was apparently in perfect health, and no antecedent morbid influence could be traced, near or remote, to which the disease could be ascribed. This was the case in 62 cases out of 114 available for comparison. In many of those in which an antecedent condition, possibly connected with the disease, was to be traced, its influence, on account of chronological remoteness and other circumstances, was very doubtful. It is probable that the largest proportion of cases in which any etiological condition can be distinctly traced is one-third. Those conditions themselves are, in various cases, of the most diverse character, and evidently constitute but a small proportion of the unknown factors.

Heredity.—The complete absence of any evidence suggesting the transmission of lymphadenosis renders it highly probable that the disease, as such, is not inherited. Almost the only facts which point to such an influence are a case recorded by Chvostek, in which the father of the patient died from hæmorrhage after the extraction of a tooth, and a case recorded by Muller, in which the patient was a man aged fifty-two, and it is stated that all his children had a tendency to enlarged glands. Nor is there much evidence of the affection of collaterals. It is possible that more careful investigation into the history of cases in patients about whose relations reliable information is to be obtained, may show that in some forms of lymphadenosis there exist relations to other forms of disease, especially to phthisis and scrofula. In a few patients there has been evidence of a strong phthisical taint, even when the gland disease possessed very distinctive features. In one case,[1] a child, aged eight years, was born six months before the death of the mother from phthisis, which existed during the pregnancy. The relations of the disease to scrofula are still more intimate. This is suggested by such examples of generalised scrofulous enlargement of glands as in the case described by Morgagni (p. 307), and by such facts as the following. In a case described by Mosler, a child, of scrofulous heredity, suffered at nine months from suppurating enlargement of cervical glands, and after measles, aet. two, presented indolent gland enlargements, of the consequences of which he died at five. The glandular disease was universal, and characteristic of this affection; there were nodules of growth in kidneys, but the spleen presented, as it often does in lymphadenosis, numbers of small spherical yellow nodules, such as are sometimes seen in tubercular disease. In several other cases the glands first affected have suppurated, those subsequently diseased presenting all the characters of lymphadenoma.

Sex.—The disease is three times as frequent in males as in females. Out of 100 cases 75 occurred in males and 25 in females. The preponderant affection of the male sex is thus greater than in splenic leucocythæmia.

Age.—The disease occurs at all ages, from one to seventy years. It is, however, more frequent in early and in late adult life. In middle life the disease is less frequent than at any other period. It has been observed in children only a few months old.[2] From the frequency of glandular enlargements in children, it might be expected to be especially common in early life; but this does not appear to be the case. Indeed, if regard is had to the larger number living, it is as infrequent in childhood as at any period of life. These inferences are based on the following table of the ages at which death occurred in 100 cases. In consequence of the difficulty of fixing the commencement of the disease in individual cases, it is only practicable to compare the ages at death:—

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[1] Recorded by Mr. Jonathan Hutchinson, Path. Trans. vol xii. p. 230.

[2] It has been asserted by Bednar that a similar condition of anæmia, glandular swelling, and enlargement of spleen, liver, and kidneys, occurs as a congenital disease, but no cases are given (Krankheiten der Neugebornen u. Säuglinge, Wien, 1850, p. 155). The occurrence of a similar condition in children at the breast, associated with leucocythæmia, has been described by Golitzinsky (Jahrbuch für Kinderheilkunde, 1861).

	Males.	Females.	Total.
Under 10 years	9	7	16
Between 10 and 20 years	12	2	14
,, 20 ,, 30 ,,	16	4	20
,, 30 ,, 40 ,,	9	5	14
,, 40 ,, 50 ,,	3	2	5
,, 50 ,, 60 ,,	18	3	21
,, 60 ,, 70 ,,	6	1	7
,, 70 ,, 80 ,,	2	1	3
Total.	75	25	100

It thus appears that one decade, between forty and fifty, presents remarkable freedom from the disease. It is more frequent under ten years of age than splenic leucocythæmia, and less frequent during middle life; for whereas two-thirds of the cases of splenic leucocythæmia occur between twenty and thirty, only two-fifths of the cases of lymphadenosis occur during the same period.

The preponderant affection of males is seen at each period of life, but varies at different epochs. It is least under ten years and at the period of life at which the disease is least frequent, viz. between forty and fifty years. At each of these periods the proportion is about as three to two; but the numbers during the latter period are too small to be relied upon. The preponderant affection of men is greatest between ten and twenty years, and between fifty and seventy; during each of these periods the proportion is as six to one.

Intemperance has appeared occasionally to have contributed to the production of the disease, but in so small a proportion of the total number of cases (7 out of 114) that its influence is manifestly slight. All of the cases were males, and most of them (five) past middle life.

Mental Depression preceded the onset of the disease in five cases; three were men and two women.

Insufficient Food in several cases was supposed to have been the cause of the disease, but in some of them it operated in conjunction with other influences. *Over-exertion* was regarded in some instances as aiding other causes, especially imperfect nourishment and exposure.

Exposure to Cold, in several cases, preceded the early symptoms of the affection. In some instances the exposure had been long continued, and had been combined with severe exertion or insufficient nourishment. In others, a single severe chill determined a series of symptoms eventuating in the glandular enlargement.

Constitutional Syphilis, from the character of the tissue changes which it produces, might be expected to be a common cause of lymphadenosis, but in only three cases was the disease ascribed to it. In one the primary disease occurred only eight months before the commencement of the glandular enlargement, and in the interval the patient had suffered from a syphilitic roseola. Iodide of potassium did not, however, hinder the rapid progress of the disease. In another the glands lessened slightly under iodide of potassium; in the third, in which primary syphilis had been contracted nine years before, antisyphilitic treatment had no effect. The relation of the disease to syphilis must therefore be considered to be doubtful.

Child-bearing.—In only one case did the disease apparently commence during pregnancy. The progress of the disease was, in another, distinctly checked during pregnancy, and it advanced rapidly after delivery. In several cases the first symptoms of the disease were observed soon after childbirth, and in one of these, recorded by Trousseau, there had been a large post-partum hæmorrhage. Two cases have been recorded by Dr. R. Paterson[1] in which very rapid and general enlargement of the glands occurred after delivery, killing the patient in a few weeks, and accompanied with extreme leucocythæmia. In one of these cases there was considerable enlargement of the spleen, and the affection appeared to have commenced during pregnancy; in the other the enlargement of the spleen was only noted during the last hours of life, the swelling of the glands was so extreme that the patient died from the asphyxia produced by the pressure of the cervical tumours.

Febrile Affections.—In very few cases has lymphadenosis succeeded a general febrile disease. One of Dr. Hodgkin's cases apparently originated in an exposure to cold during the convalescence from a fever. In some instances (5 out of 114) the patients had suffered previously from intermittent fever. In one patient the disease succeeded hooping cough.

Local Irritation.—It was pointed out by Craigie and by Trousseau that in some cases a local irritation had appeared to be the exciting cause of the glandular swellings. It has already been mentioned that in some cases local glandular enlargement has preceded, for a time, the general affection. In some of these patients the local glandular swellings could be attributed to no exciting cause, but in others they were apparently due to a local irritation. The irritation of a decayed tooth, of chronic coryza, and of discharge from the ear, appeared, in three cases respectively, to be the causes of an initial enlargement of the cervical glands. In three other cases a sore throat appeared to produce the same effect. In one, for instance, a catarrhal tonsillitis was followed by considerable and permanent enlargement of one tonsil, and subsequently, of the lymphatic glands of the neck. The sore throat, in one case, was syphilitic in character. The disease commenced, in two cases, by enlargement of the glands secondary to an inflammation of the lachrymal sac, and in one of these the glands first enlarged suppurated. Dr. Wilks has recorded a case in which the disease commenced by enlargement of the glands of the groin, apparently excited by a soft chancre. General eczema, in another patient, preceded a generalised onset of the affection. There is thus reason to attribute some effect in the production of the disease to such irritation as does, not uncommonly, set up simple glandular enlargements. The significance of this will be discussed in the section on Pathology.

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[1] Edin. Med. Journal, 1870, p. 1074.

PATHOLOGICAL ANATOMY.—In all cases of lymphadenosis, with the exception of certain very rare forms of visceral growth, there is found enlargement of some of the lymphatic glands; in most, there is also enlargement of the spleen; and in a considerable number there are lymphatic growths, *i.e.* growths of adenoid tissue, in other situations, in the alimentary canal, in the kidneys, in the liver, in the medulla of bone, and occasionally in the lungs, in the testicles, and in the heart.

LYMPHATIC GLANDS.—The lymphatic glands are arranged in groups throughout the body, some superficial, others deeply seated within the visceral cavities. Many, often all, the glands of the same group are diseased. The affection may be confined to one or two groups; more commonly it involves several groups, those which are deeply seated in the visceral cavities as well as those which are superficial. Not unfrequently all the glands, deep and superficial, are diseased. The several groups of glands are affected in the following order of frequency, the first being those most frequently diseased:—

(1) Cervical; (2) Axillary; (3) Inguinal; (4) Retroperitoneal; (5) Bronchial; (6) Mediastinal; (7) Mesenteric.

Certain subsidiary groups of glands are often diseased in association with those adjacent, as the sub-maxillary and occipital with the cervical, the epitrochlear with the axillary, the popliteal with the inguinal. Nodular growths, precisely resembling enlarged lymphatic glands, may he found in situations in which the existence of glands is not usually recognised. These arise either from the enlargement of small glandular structures which, in the normal state, pass unnoticed, or from the development of adenoid tissue along the course of the lymphatic vessels. Such are the chains of growths often observed beneath the pectoral muscle extending upwards from the axilla to the neck, or inwards to the mediastinum. In most cases the glands are enlarged on both sides, but the same group may be affected on one side only, or on the two sides in very different degree.

The size attained by the individual glands varies from that of a nut to that of a pigeon's egg, or even a hen's egg, while the mass formed by the enlargement of a whole group may reach a considerable size, equalling that of a child's head, in either the neck, axilla, thorax, or abdomen. In one case the whole of the enlarged glands weighed ten pounds. In shape the individual glands are oval or rounded, sometimes flattened by pressure. At first they are separate, and freely movable one on another. Commonly, after a time, the glands unite to form a conglomerate mass in which the several glands are no longer movable. Sometimes glands in one group, which is the largest, are thus fixed, while those of other groups remain movable. This adhesion of the glands to one another is usually a consequence of the perforation of their capsule by the growth which causes their enlargement, and its extension from one gland to another; more rarely it is due to a process of peri-adenitis, involving the tissues adjacent. When the growth has thus passed beyond the limit of the gland itself it may extend in the adjacent connective tissue, invade a contiguous organ, or perforate a large vessel. It is rare for the skin over superficial glands to be affected, but occasionally it becomes involved in this manner. The proper elements of the tissues thus invaded undergo atrophy, fibrous structures are infiltrated and split up by the lymphoid cells, epithelium and muscular tissue degenerate. The enlarged glands often exert pressure on adjacent parts, may displace organs, and interfere seriously with hollow viscera.

Affection of the several Groups of Glands.—Although the groups of glands are named according to their position, it must be remembered that, as already stated, there are outlying groups of glands which are included in the several groups, and that between the several groups continuous chains of growths may exist.

Cervical Glands.—The group of glands in the posterior triangle, above the clavicle, is generally affected, and may form a mass of very large size. Those in the upper part of the neck, along the sterno-mastoid muscle, are also enlarged, and frequently those which lie beneath the lower jaw, submaxillary glands; and at the back of the neck, occipital glands. The submaxillary glands may meet in the middle line, and encircle the neck below the jaw. A chain of enlarged glands may extend, upwards, along the internal jugular vein to the base of the skull; downwards, beneath the clavicle and pectoral muscle, to the axilla; and also along the trachea into the thorax, where they may be continuous with the mediastinal or bronchial glands. A large growth from the lower cervical glands may descend in front of the clavicle and lie over the upper part of the thorax. The cervical tumours may exercise serious pressure on the structures in the neck. If the enlargement is much greater on one side than on the other, the larynx may be displaced. The pressure on the trachea may interfere with respiration, and even cause death by suffocation. This less frequently results from pressure in the neck than from the pressure of the thoracic glands on the lower part of the trachea. The enlarged glands in the neck may press on the recurrent laryngeal and cause paralysis of the larynx. Those more deeply seated may compress the internal jugular vein and cause thrombosis in it. When the growth is invasive in its character, it may infiltrate the wall of the vein.[1]

Axillary Glands.—These are frequently diseased and often attain a very considerable size; they may weigh one or two pounds. Those which lie in the anterior part of the axilla often extend beneath the pectoral muscle by finger-like prolongations. Occasionally the epitrochlear glands above the elbow are also diseased. The glands in both axillæ are usually enlarged, but they are always larger on one side than on the other. There is no regularity in the order of these glandular enlargements. Sometimes the glands in the neck and axilla are largest on the same side; but in other cases the glands on one side of the neck and in the opposite axilla may be enlarged first and chiefly.

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[1] Schultz, Arch. der Heilkunde, 1874, Case 5.

Thoracic Glands.—Those in the anterior mediastinum are often enlarged, and, not unfrequently, the disease commences in, and may be limited to, this group. The growth here often extends beyond the glands and invades adjacent structures. The thymus may be thus invaded. Occasionally the growth begins in the thymus, and the tumour produced is, according to Virchow, more uniform and less nodular than that which results from primary enlargement of the glands. Sometimes the thymus remains free, and has been found, in children especially, in a normal state, although surrounded by masses of enlarged glands. In other cases the growth extends in the cellular tissue of the anterior mediastinum, passing between the sternum and the pericardium, and reaching from the highest part of the pericardium to the diaphragm. When the enlargement is considerable, the heart and left lung are frequently displaced. The growth may be closely connected with the sternum, and may perforate the pericardium. Sometimes the pericardium is not invaded although the growth spreads widely in its vicinity.

The glands which lie beneath the arch of the aorta, and have been termed the *cardiac* glands, are sometimes diseased, and much more frequently the *bronchial* glands, which are adjacent to the bifurcation of the trachea, the bronchi, and the roots of the lungs. These are numerous, and, by their enlargement, constitute frequently a mass of considerable size, which may be continuous above, along the trachea, with the cervical glands. The trachea and bronchi are frequently pressed upon, and may be so narrowed as to interfere with the passage of air. Sometimes the trachea, although surrounded by growth, is not compressed. Trousseau has pointed out that the great vessels rarely suffer from compression. The growth here is frequently invasive, and may perforate the posterior surface of the pericardium, often in many places. The perforating growth may have a smooth concave surface within the pericardial sac to correspond with the adjacent portion of the heart.

Frequently the growth from the bronchial glands extends on each side into the lungs, passing into them around the bronchial tubes. Occasionally the bronchial glands escape, even when those of the anterior mediastinum are extensively diseased. The glands which lie in the posterior mediastinum, around the aorta and œsophagus, may be enlarged and connected in front with those around the bronchi, and above with those adjacent to the pharynx. They do not often compress the aorta or œsophagus, or the thoracic duct. The pressure in the azygos veins has caused thrombosis in them, with extensive pleural effusion.[1] Occasionally, in those forms in which the growth spreads widely, the vertebræ may be invaded.

Abdominal Glands.—The retro-peritoneal glands, which lie along the spine beside the aorta and vena cava, are most frequently affected, and often form a mass of large size, which may be continuous above with the glands in the thorax, and below may extend along the iliac vessels and down into the pelvis. In a case recorded by Bonfils the pelvic and lumbar glands together weighed eight pounds. These glands rarely exercise much pressure, but those within the pelvis have been known to press on a ureter and cause its dilatation above the part compressed. Occasionally the retro-peritoneal glands in front of the spine surround and compress the solar plexus. When this is the case there may be bronzing of the skin although the supra-renal bodies are healthy. Some years ago I made a post-mortem examination on the body of a patient of Sir William Jenner in which this was the case. The glandular enlargement was general, but in the thoracic and abdominal cavities it was very great. The supra-renal body on each side was healthy, but the nerves from each passed into the mass of gland growth by which the solar plexus was inclosed. The bronzing of the skin was striking, and had the distribution characteristic of Addison's disease. A very similar case has been recorded by Féréol.[2] Other instances of discoloration of the skin will be mentioned in speaking of its changes.

The *Mesenteric* glands are sometimes enlarged, but far less frequently than the retro-peritoneal group, and less frequently, indeed, than any others. When diseased, the size they attain is commonly moderate, the individual glands rarely exceeding that of a pigeon's egg. They are often continuous with a chain of glands which extend up along the gastro-hepatic omentum to the liver—portal glands. These may compress the portal vessels and the bile duct. The glands in the gastro-splenic omentum and the hilus of the spleen may also be enlarged. The size attained by these visceral glands is sometimes considerable. In one case the portal glands formed a mass the size of a child's head.[3] The glands about the pancreas are sometimes diseased, and so also are those of the meso-colon.

The *Inguinal Glands* are affected in about half the cases. They often attain a large size, and may be continuous beneath Poupart's ligament with the iliac glands. The femoral vessels and crural nerves may be compressed. Other enlarged glands or gland-growths may extend down the thigh along the femoral vessels. The popliteal glands are also sometimes enlarged, but rarely to a considerable extent.

Characters of the Glandular Enlargement.—The physical characters of the glands depend on the rapidity and duration of the enlargement, and on the characters of the growth causing it. The consistence of the glands varies. In some cases they are soft; in others of semi-cartilaginous hardness. Virchow distinguished between these forms. In some cases, however, the characters of the enlargement are not uniform: the large glands may be soft and the smaller firm and elastic. Or the converse relation may be observed, the small glands may be soft and the larger hard. In other cases, the more deeply-seated glands may be soft and fleshy, while the superficial glands are tolerably firm. Enlarged glands may be at first soft, and afterwards become much harder. The capsule is often normal in thickness, now and then it is thickened by a process of peri-adenitis, and sometimes it is perforated by growth from the interior of the gland.

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[1] Dr. J. J. Black, American Journal of Medical Science, April, 1868.

[2] Féréol and Nieszkowski, Gaz. des Hôp. 1867.

[3] Desnos et Barié, Gaz. Méd. de Paris, 1876, Nos. 34 and 35.

The appearance of a section varies considerably. It is usually more or less uniform, the normal distinction between cortex and medulla having disappeared. The colour is whitish or yellowish-grey, mottled with red in proportion to the vascularity of the gland. Sometimes the follicles have a different appearance from the rest of the gland, either due to a thickening of the septa between them, or in consequence of the degeneration of their contents, which then are of a yellow colour and contrast with the grey tint of the rest. Rarely, the fibroid change which sometimes occurs is more advanced in the central than in the cortical portion of the gland, and then a distinction between the two parts is to be observed. Often irregular tracts of dense fibrous tissue pass in various directions. The glands do not usually present any fatty degeneration such as is so common in scrofulous affections. Here and there a gland may be found which has undergone caseation, wholly or in part. In one case, for instance, a gland in the groin was cheesy and softened, while all the other glands were firm, tough, and translucent. Occasionally many glands present this change, but such cases must be regarded as occupying an intermediate position between lymphadenosis and scrofula.

The softer glands yield juice on scraping. The firmer glands yield no juice. The harder glands are very firm and resist the knife. The section has a shining close aspect. This condition is common in cases of long duration and slow course.

When the capsule has been perforated, the gland presents a bulging, and at the bulged portions the tint is grey, and the capsule is obviously thinned or destroyed. The growth having perforated the capsule, invades any tissue or organ which may be adjacent.

Microscopical Structure.—The histological changes in the glands vary in different cases. In the smaller and softer glands the only recognisable change is an enormous increase in the cellular elements, the lymph corpuscles which lie in the meshes of the reticulum. The normal relations of structure, the relations between the septa and the follicles, and between the cortical and medullary substance of the gland, remain unchanged. Sometimes the growth of cells gradually invades the septa, which become split up and disappear. The substance of the gland then presents on section a uniform appearance. All parts present the structure usually seen within the follicles; leucocyte-like cells lie embedded in a moderately close network of interlacing delicate fibres and films, provided here and there with nuclei. This stroma can only be well seen on pencilling a section. Schultz believes that the delicate films of the stroma arise from the septa of the normal gland, split up and separated in various directions by the growth and accumulation of cells. Towards the hilus, where the vessels are larger, an infiltration of their outer coat with similar cells may be traced, and this infiltration may pass along the vessels to the exterior of the gland, and there extend in the adjacent fatty and cellular tissues. The new growth perforates the capsule by a process of progressive cellular infiltration, similar to that by which it invades the interfollicular septa.

The cells are round bodies, usually with single-nucleolated nuclei, and resemble very closely ordinary lymph cells. Sometimes larger cells are seen. Occasionally, in the soft forms, a multiplication of the nuclei takes place, and many-nucleated cells and even giant cells may be found, resembling those seen in myeloid tumours (Schmuziger). Similar cells may even be seen in harder forms (Langhans). Ollivier and Ranvier assert that some of these larger cells provided with one or two nuclei possess a diffuse colouring matter.

In the glands which are firmer there is a larger amount of fibroid tissue. In some cases this is mainly confined to the interfollicular septa, but it usually involves also the reticulum, the meshes of which become greatly increased in thickness: they may be homogeneous or have an obscurely fibrous aspect. They may be so abundant as to separate the individual cells. The thickening of the reticulum is greatest in the neighbourhood of the vessels, both large and small. The capillaries may have their walls considerably thickened, and have a homogeneous or longitudinally striated appearance. The septa then cease to be distinguishable from the follicles. Ultimately many cells perish, and the fibroid transformation may be so complete that very few cells are to be seen on section of the gland, only here and there a group or series of small round cells lying between the fibres. The latter may be broad, wavy, and present a hyaline or "vitreous" appearance. These vitreous fibres may be arranged concentrically around the vessels.

In addition to the nuclei at the junction of the fibres of the stroma, fusiform cells are sometimes seen mingled with the reticulum. According to Virchow, round granular bodies may occasionally be found which are globes of nuclei of epithelial appearance, sometimes with many-nucleated giant cells. It is probable that these arise in the same manner as that which the writer has traced in the growth of "nested sarcomas."[1] Virchow has remarked that in one case of hard glandular enlargements, the vessels presented an amyloid degeneration of the walls.[2] There are few instances of amyloid degeneration of the glands on record.

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[1] Med. Chir. Trans. 1876.

[2] Krankhaften Geschwülste, vol. ii. p. 618.

The histological varieties may be thus summarised :—

1. A soft cellular growth of early and rapid enlargement, breaking up the septa by infiltration, and converting the gland into a uniform soft cellular mass, in which no distinction of cortical and medullary substance is perceptible.

2. A mixed cellular and fibrous change in which the cells are still abundant, but the reticulum is also thicker, and the septa, instead of being broken up, are also thicker than normal. The cells between the fibres are apt to undergo degeneration.

3. A more advanced fibroid change in which few cells are visible, but the whole gland is transformed into a dense network of broad fibres more or less uniform in appearance, with a thickened capsule.

Lymphatic Vessels.—There is little direct evidence of the implication of the lymphatic vessels in the morbid process. Those of the mesentery were observed in one case to be dilated. It is possible that the growth may sometimes spread along these vessels, since in other organs there is strong evidence that it may be located in structures with which the lymphatic vessels are connected. Hodgkin remarked that in one case the absorbents connected with some of the glands appeared to contain a bloody fluid. Such an appearance may have been produced by hæmorrhage into the gland. Wunderlich found the lymphatic vessels from a diseased gland enlarged and distended with a clear fluid. In other cases, however, all attempts to discover or inject the lymphatics of the glands have been fruitless, and it is assumed that they may have been destroyed.

ORGANS.—*Spleen.*—In a large proportion of the cases the spleen is diseased. Out of ninety-five post-mortem examinations in which the state of the spleen is mentioned, it is said to have been normal in about one-fifth (twenty cases), while in the remaining seventy-five cases it was more or less diseased, in almost all of them enlarged. The degree of enlargement varies in different cases, being in some instances slight, in most moderate, in a few great. The organ attains a large size much less frequently than in splenic leucocythæmia. It seldom exceeds ten inches in length, and is more frequently from six to eigh inches long, the average length of the healthy spleen being taken at five inches. The weight of the spleen is, as a rule, increased in proportion to the increase in size, and reaches from 10 to 30 oz., rarely exceeding the latter. In a few cases the spleen has reached an extreme size.

The cause of the enlargement is, in a minority of the cases, a "simple hypertrophy," similar to that found in splenic leucocythæmia. In the majority it is due to the presence of disseminated growths, in the form of minute spots, or larger nodules and masses. Out of the seventy-five cases in which the spleen was enlarged, growths in the organ existed in fifty-six: in the remaining nineteen cases the organ was stated to be simply hypertrophied, or else to be "enlarged," any special cause of enlargement not being mentioned.

The enlargement of the organ due to the disseminated growths is rarely extreme. In some it was slight, and in a few, growths existed in a spleen of normal size.[1] In a few the enlargement was very great. In the majority of the cases in which the weight is given it has varied between one and two pounds. In only a few cases was this weight exceeded, and in two of these an enormous size was attained, the spleen weighing respectively seven[2] and nine-and-a-half[3] pounds. The surface may be uneven from the projection of the nodules of growth. The growths are in some cases small in size, visible as round pale spots, the size of peas, in the darker splenic pulp, and apparently seated in the Malpighian bodies. In most cases the growths are larger and more or less irregular in shape, frequently angular, and attaining the size of a pea, a hazel-nut, or walnut. The growths are greyish-white or yellowish-white, and more or less opaque. They were compared by Dr. Hodgkin to masses of suet, by others to bacon-fat run into the organ. They are commonly not surrounded by any capsule, but around the larger growths the splenic tissue may be compressed. They are usually firmer than the splenic tissue, and are sometimes very hard. Their consistence may correspond with that of the glands in the same case. These angular masses of growth rarely present softening or degeneration, unless many nodules are aggregated together into a large mass, when some local softening may occur. The spleen sometimes contains small round nodules the size of peas, more opaque and yellow than the irregular growths, and these occasionally soften. The spleen then resembles very closely the appearance sometimes presented in tuberculous disease. The growths have been observed, although very rarely, to invade the capsule. The splenic pulp may be normal in quantity, or may be atrophied and reduced to narrow tracts between the nodules of growth.

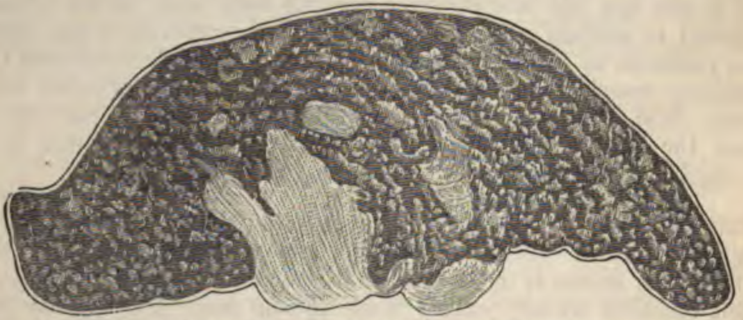

FIG. 1.—Spleen in Hodgkin's Disease, after Virchow.

The spleen, when simply hypertrophied in this disease, attains on an average a somewhat larger size than when it is the seat of the disseminated growths. Its consistence varies: it is usually firm, sometimes hard, rarely soft. The Malpighian bodies are often conspicuous, especially in the slighter degrees of enlargement, when they are often slightly enlarged and visible as paler grey spots. It is probable that these are the early stage of the disseminated growths, for between the two, in different cases, every gradation may be traced.

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[1] In a case recorded by Dr. Strangeways Hounsell, of general gland disease in a lad of 14, the spleen contained nodules, and weighed only 3½ ozs. But the case was, in some respects, closely allied to scrofulous disease.

[2] Schultz, loc. cit. Case 6.

[3] Wunderlich, Arch. der Heilkunde, 1866, p. 535.

Besides the disseminated angular growths, wedge-shaped infarctions may be observed, similar to those seen so often in leucocythæmia. They are commonly situated on the surface, the base of the wedge being adjacent to the capsule. When recent they are red, often the seat of extravasation, and surrounded by a zone of intense congestion. When of older date, they are paler red, granular in aspect, and ultimately become yellowish-white and opaque. The resemblance in the naked eye characters between the angular growths and these areas of infarction is close, and each condition has been repeatedly mistaken for the other. These infarctions are distinguished from the larger masses of growth by the congestion in the early stage, by their situation, the relation to the surface, and their greater opacity.

The spleen, enlarged from whatever cause, frequently contracts adhesions to adjacent organs, especially to the diaphragm and liver. The capsule is also often thickened by the same chronic inflammation which causes the adhesions.

Microscopical Structure.—The growths in the spleen resemble closely the growth in the lymphatic glands. They consist of the same minute cells lying in the meshes of a reticulum, identical in appearance with that of the lymphatic glands. The large, many-nucleated cells found in the glands, are rarely seen in the spleen.[1] There is often an abundance of imperfectly formed fibrous tissue, forming thick vitreous-like bands. Spindle-shaped cells connected with the reticulum are not infrequent. In the smaller growths the new tissue can often be seen to be connected with the walls of the vessels, and to spread along them. The limitation of the growths in the Malpighian bodies from the adjacent splenic pulp is, under the microscope, less distinct than it appears to the naked eye, but may commonly be brought out sharply by staining, and recognised by the relation to the vessels. Sometimes the tissue of the pulp is compressed in the vicinity of the growth, and may contain much brownish-red pigment. The trabeculæ of the spleen may be infiltrated with lymphoid cells, which may also invade the walls of the vessels, especially the veins. The structure of the pulp is otherwise normal; it may present an appearance of atrophy from compression; the cells may be scanty, and the trabeculæ thickened, or it may present the same over-growth seen in splenic leucocythæmia. In one case recorded by Schmuziger, there appeared to be an enormous increase in the trabeculæ of the pulp.

Lardaceous degeneration is rarely observed in the spleen. In one case I found this degeneration in the Malpighian bodies, and not in the glands. In another case some of the smaller growths in the spleen, evidently originating in the Malpighian follicles, presented this degeneration, while the larger growths did not. A similar degeneration in the vessels of the spleen was found by Huttenbrenner in a case in which it was also present in the vessels of the glands and kidneys.

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[1] An instance of their occurrence is recorded by Langhans. Virchow's Archiv, vol. liv. 1872, p. 513.

The medulla of bones may present a change similar to that met with in some cases of splenic leucocythæmia and pernicious anæmia. Softening of the bones was noticed in this affection by Perrin,[1] as long ago as 1861, in a case in which the ribs could be cut through with a scalpel, and the least pressure caused their fracture. The marrow is converted into a reddish-grey semi-diffluent substance. The change has been observed in the ribs, sternum, vertebræ, femur, tibiæ, and cranial bones. It has been found in cases in which there was a slight excess of pale corpuscles in the blood, and also in cases in which there was no such excess. It is not possible at present to say with what frequency the medulla is affected. It has been examined in several cases and found normal.

Under the microscope the altered medulla presents abundant lymphoid cells, and rather larger cells with very large nucleolated nuclei. In a section of bone, hardened and decalcified (with picric or chromic acid), these cells are seen to cram the enlarged spaces of the bone, and by pencilling, a reticulum may be traced, the trabeculæ of which are connected with the walls of capillaries (Kelsch.) Thus there is a substitution of adenoid tissue for the normal elements of the medulla.

Alimentary Canal.—The numerous minute collections of adenoid tissue in the alimentary canal very frequently become the seats of morbid growths of similar tissue in cases in which the affection of the glands is widely spread, and occasionally in cases in which the glandular disease is slight. The mucous membrane of the mouth and the gums may present traces of inflammation, swelling, and ulceration, especially in the later stages of the disease. In the follicles on the posterior part of the tongue there may be a considerable overgrowth of tissue, rendering them unduly prominent.

The tonsils are occasionally enlarged by a similar growth of the adenoid tissue, which composes so large a proportion of their mass. The enlargement commonly affects both tonsils, less frequently one only. Occasionally the tissue may degenerate, and ulceration occur. Such ulceration is usually unilateral. One tonsil has been observed to be of an enormous size, and the other to have ulcerated away.[2] A lymphoid growth occupied the soft palate in a case recorded by Mulder.

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[1] Perrin, Bull. de la Soc. Anatomique, 1861, p. 247. Other cases are recorded by Kottmann, Inaug. Diss. Bern. 1871, and Schmidt's Jahrbuch, 1872, vol. clv. p. 327 (Case 4); Kelsch, Bull. de la Soc. Anat. 1873; Ponfick, Virchow's Archiv, vol. l. p. 550; Schultz, Archiv der Heilkunde, 1874, p. 200 (Case 1); Schmuziger, ibid. 1876, p. 279.

[2] Wickham Legg, St. Bartholomew's Hosp. Reports, vol. xi. p. 70.

Glandular overgrowth may occur in the mucous membrane of the pharynx. Occasionally, behind the pharynx, extensive growths occur, which may involve the pharyngeal wall and cause degeneration of its muscular fibres.

Growths in the œsophagus have been observed in several cases. The mucous membrane has been found thickly studded with minute new formations of lymphatic tissue, each about the size of a poppy-seed.[1] In one case there was extensive infiltration of the mucous membrane by a similar growth, in places breaking down into ulcers, and closely resembling that which is occasionally seen in the stomach.[2]

In the stomach, the lymphoid tissue, which exists beneath and between the glands of the mucous membrane, sometimes undergoes extensive hyperplasia, so as to cause irregular thickening of the mucous membrane, even over its entire surface. In a case described by Cruveilhier these growths constituted large thick rolls, resembling in size the cerebral convolutions. The growth here is always soft, and on section yields an abundant juice. It may break down and ulcerate, causing many small round ulcers.

When there is an extensive development of adenoid growth in the stomach, the upper part of the duodenum is usually affected in a similar manner.

Throughout the intestines the collections of lymphoid tissue in the solitary glands and Peyer's patches often undergo great enlargement from the growth of similar tissue. The enlargement of both sets of glands may be most considerable, as in typhoid fever, in the lower part of the ileum, and may extend into the commencement of the ascending colon. Occasionally the solitary follicles are alone considerably enlarged throughout the small and large intestines.

The growth of adenoid tissue, commencing in the follicular glands, often extends beyond their limits, and then its original relation to the glands may be obscured. Schultz indeed asserts that the growth is entirely a new formation, before which the original lymphatic tissue perishes, and that the new and the old can always be distinguished in the early stage of the growth by the deeper staining with carmine of the new portion. The growth may extend from the lymphatic follicles widely in the mucous tissue. Occasionally nodules appear in the mucous membrane unconnected with the follicular glands. The growth remains limited to the inner layer of the wall of the intestine, never invading the muscular coat. Schultz has observed that it may perforate the delicate layer of muscular fibres which exists beneath the glands, and may invade the villi of the intestine, spreading there, as elsewhere, along the outer coat of the arteries. The Lieberkühnian crypts may be pressed upon, and their epithelium undergo degeneration. The growth occasionally assumes a very extensive development, and leads to great thickening of the wall of the intestine, which does not however usually reduce the calibre of the bowel, a point to which

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[1] Chvostek, Allg. Wien. Med. Zeitung, 1877.

[2] Greenfield, Path. Trans. 1876, p. 275.

Dr. Moxon has called especial attention.[1] A general infiltration of the wall, in a case recorded by Kelsch,[2] attained in places such a local development as to constitute actual tumours. Irregular ulcers had formed, one of which surrounded the intestine.

The mucous membrane of the rectum may be the seat of adenoid growth very similar to that seen in the stomach.

Liver.—In a considerable number of cases the liver is diseased. Increase in size has been observed in about one-quarter of the cases, but this does not represent the frequency with which the liver is affected. The most common change is the existence of scattered lymphoid growths, and these may exist without causing enlargement, and may not be discoverable on naked-eye examination. The growths are usually minute, varying in size from a pin's head to a small pea, and are greyish in tint. Their distinctness depends on their colour and on the tint of the hepatic tissue.

Less commonly, nodules are met with of larger size and fewer in number, varying from the dimensions of a large pea to that of a hazelnut or a bean, white and semi-opaque in appearance, resembling very closely those of the spleen. They are usually firmer than the smaller growths, and are occasionally very hard, even semi-cartilaginous in consistence. They are sometimes rounded, but more often irregular in outline.

Under the microscope the minute growths are seen to occupy, as a rule, the interlobular spaces in which the portal vessels lie, and thence they may send processes into the interior of the acini. In structure they consist of minute round cells, lying in a retiform stroma, such as constitute the lymphatic tissue elsewhere. According to Schultz the trabeculæ of the tissue can be resolved into delicate membranous films, nucleated, and often provided with ramifying stellate cells. In one case many spindle cells were seen in the hepatic growths, although those in the glands and spleen had the characteristic retiform stroma.[3] The liver-cells do not participate in the process, but perish before it, the acini being invaded by processes of growth which develope between the rows of liver-cells. Occasionally a layer of compressed liver-cells exists on the outer surface of the growth, and constitutes a sort of capsule. When the nodules are of large size and irregular, tracts of atrophied acini may intervene between them. The cells of the growth may infiltrate the walls of the portal veins, penetrating even to the intima, which may, according to Schultz, be transformed into a layer of spindle cells. The blood thus appears to pass in channels excavated in the new growth. A similar but slighter infiltration of the walls of the arteries may be seen, and more frequently still of the walls of the bile ducts.

In some cases, instead of disseminated nodules, a diffused growth occupies all the portal interlobular spaces, and is visible to the naked eye as fine white lines accompanying the portal vessels, and under the microscope as broad tracts of nucleated tissue. Sometimes minute nodules may be observed here and there. From these tracts of tissue extensions may take place into the substance of the acini between the rows of liver-cells, just as from the nodular growths. In a few instances a similar growth has been seen in the neighbourhood of the hepatic vein. This diffused growth may be so abundant as to occupy, it has been estimated, one-third of the organ.[1]

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[1] Path. Trans. 1873.

[2] Bull. de la Soc. Anatom. 1873.

[3] Payne, Path. Trans. vol. xiv. 401.

Ranvier has pointed out that in some cases in which the liver is enlarged there are no growths, but merely a great distension of the capillaries with blood, even when the latter contains no excess of leucocytes. The two conditions of congestion and growth are occasionally conjoined, as in an instance related by Bohn,[2] in which the liver was enormously enlarged from passive congestion, and throughout were scattered numerous foci of lymphatic growth.

The new formations rarely exhibit degeneration. The increased consistence depends on a thickening of the retiform tissue, similar to that which occurs in the glands. Fatty degeneration in the centre of the new growths has, however, been occasionally observed. Lardaceous degeneration of the walls of the portal venules was found in one case by Schmuziger.

The liver tissue itself may be the seat of fatty degeneration, which, in several cases, has been very intense.

The *Pancreas* rarely contains lymphatic growths. In a case recorded by Dr. Caton [3] a small tumour of adenoid structure projected from the anterior surface.

Kidneys.—Adenoid growths, similar to those which occur in the liver, are met with in the kidney in a considerable number of cases. They are mentioned in fifteen autopsies out of seventy, but were probably present in a much larger number, since they are rarely conspicuous, and sometimes can be detected only by microscopical examination. The organs thus affected may be of normal size or enlarged. They are usually unduly pale, and the pallor and increased size are chiefly due to the state of the cortical portion. When the growths are visible to the naked eye, they usually vary in size from that of a mustard-seed to that of a pea, occasionally, but very rarely, attaining the size of a hazel-nut. In one case a kidney was the seat of a very large lymphomatous growth.[4] They are always pale, whitish-grey in tint, and when large are usually firmer than the kidney substance in which they lie.

The growths are most abundant in the cortex. They commence in the intertubular tissue, and consist of the same retiform stroma and corpuscles as have been already described. By their enlargement they compress the tubules, and may lead to atrophy of the epithelium at the part compressed. Pale wedge-shaped areas, due to infarction from vascular obstruction, may also be occasionally found.

The substance of the kidneys may be affected independently of the existence of growths. The most common appearance is that of parenchymatous degeneration, showing itself either as cloudy swelling or as the mottled or large white kidney. Amyloid degeneration was present in one case.[1] Uric acid concretions have been observed in a few cases.

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[1] Wilks, Guy's Hosp. Reports, 1866, p. 61.

[2] Deut. Arch für Klin. Med. 1869, p. 429.

[3] British Med. Journal, August 20, 1870.

[4] Desnos and Bariè, loc. cit.

The *Peritoneum* has been found inflamed over enlarged glands. In a few cases it has been the seat of lymphoid growths. These sometimes closely resemble tubercle, and have been regarded as such. But sometimes they are as large as peas, and consist of the characteristic retiform tissue. In a case recorded by Bohn such growths existed in the peritoneum over the liver and intestine; and in another case similar nodules were scattered over the surface of the stomach and colon. The omentum has also been observed to be set through with small nodules, which Langhans in one case found to surround the vessels, and to possess a structure identical with that of the glands. They were bounded by bundles of connective tissue.

The peritoneal cavity frequently contains fluid, as part of general dropsy, in consequence of lymphatic growth in the hilus of the liver obstructing the portal vein, or, less frequently, in consequence of the direct irritating influence of the process of lymphatic growth in the peritoneum.

The *Ovaries* are very rarely diseased. In one case a growth of considerable size was found in one ovary. In another case, beneath the capsules of the ovaries, were minute nodules, having a lymphoid structure, and precisely resembling similar growths in the lungs.[2]

Testicles.—Growths have been met with in the testicles in many cases, usually in one, and sometimes in both. They may begin in the epididymis and be confined to it, as in one of Dr. Hodgkin's cases, or they may invade the secreting substance of the organ. In one case, recorded by Schultz, without leucocytal excess, both testicles were enlarged to double the normal size, the enlargement being due to infiltration with soft growth, occupying about two-thirds of each organ.

The structure of the growth in the testicle is the same as that met with elsewhere. It has been traced by Schultz to pass from the corpus Highmori into the septula, and between the seminal tubules along the blood-vessels. The walls of the vessels gradually become changed and invaded by the growth, so that at last they cannot be recognised, and the blood seems to circulate in wall-less channels in the lymphoid tissue. The seminal tubules are pressed upon, and their walls invaded by the growth, the epithelium within them undergoing degeneration. When the growth reaches the tunica albuginea, the same process can be traced which has been described as occurring in the capsule of the lymphatic glands. The new growth contains many newly-formed vessels.

Supra-renal Bodies.—In a man, aged twenty-three, who died in University College Hospital (under the care of Dr. Wilson Fox), the glandular enlargement being general and great, the supra-renal bodies were large, firm on section, the medullary part was hard and light-coloured, and in some places nodulated in the same manner as the rapidly growing lymphatic glands.

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[1] Schmuziger, loc. cit. p. 27.

[2] Hérard, L'Un. Méd. 1865, Nos. 90 and 91.

The *Thymus* gland is frequently involved, as already stated, by growth from glands in the anterior mediastinum. It is sometimes independently affected, and the growth from it may invade adjacent parts. The thymus has also been found to be enlarged and separate from the enlarged glands.

The *Trachea* is not often diseased. In one case the mucous membrane was covered with minute growths the size of poppy-seeds. (Chvostek.)

The *Lungs* may be invaded directly by the growths in the bronchial glands, or may be the seat of disseminated new formations similar to those which are met with in other organs. The invasion of the root of the lung from the glands takes place around the bronchi and around the vessels, and also, in some instances, by direct extension. The peri-bronchial connective tissue is believed to be part of the lymphatic system. The growth forms nodules, varying in size from a pea to a walnut, sharply limited from the lung tissue, greyish-white in colour, and resembling exactly the appearance of the glands themselves.

The disseminated growths which occur through the lung substance are pale, translucent or whitish grey, the size of mustard-seeds or larger, and are often mistaken for tubercles. They are commonly most abundant towards the hilus, especially when of some size. In other cases they are uniformly scattered, or may occur chiefly towards the base, or towards the surface of the lung. Some of these growths are surrounded by a zone of congestion, probably from the collateral congestion due to the destruction of some of the vessels. The adjacent congestion may go on to actual pneumonia. The growths rarely break down, certainly less frequently than do the growths which occur in leucocythæmia. In one case on record masses were found in both lungs, and in the upper lobe of one were cavities containing pus, but whether formed by the breaking down of growths was uncertain.[1] The growths are composed of the same lymphatic tissue as is met with elsewhere, and they commonly develope in the tissue which surrounds the smaller bronchi. In some cases true tubercle has been found in the lungs.

The lungs are rarely the seat of other parenchymatous changes. That which is most commonly found is œdema, occurring as part of general anasarca, and it is a not uncommon cause of death. Occasionally they present the signs of acute pneumonia. Rarely there is evidence of chronic pneumonia, such as occurs in phthisis, apical, caseating.

Pleura.—Small sub-pleural lymphoid growths are found in this disease, although rarely. Effusions of fluid may occur into one or both pleural cavities, commonly serous, but sometimes sanguinolent.

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[1] Löschner, Aus dem Franz-Josef Kinderspitale in Prag, Theil ii. 1868, p. 237.

Nodules of lymphoid growth have been occasionally observed in the substance of the diaphragm.[1]

The *Heart* is sometimes normal, but frequently it is small. Occasionally it exhibits intense fatty degeneration, as in other forms of extreme anæmia. In a few cases lymphoid growths have been observed in the substance or on the surface of the heart,[2] and in one case numerous growths existed beneath the endo- and peri-cardium. A growth originating in the thymus may extend over the whole anterior surface of the pericardium, and almost envelope the heart. Growths in the mediastinum may perforate and shape themselves to the contiguous part of the heart. Effusion into the pericardial sac is not unfrequent, and, like that in the pleura, is sometimes sanguinolent.

The *Brain* is rarely the seat of growths in these cases, although the structures about the brain occasionally present primary lymphadenoma. In a case recorded by Dr Murchison, a small growth existed in the dura mater above the foramen magnum,[3] and small lymphoid tumours were found, in another case, above the foramen opticum.[4]

Retina.—In some cases of Hodgkin's disease, with excess of white corpuscles in the blood, changes in the retina have been observed similar to those in leucocythæmia splenica. They have not, however, been described in any case in which there was no leucocytal excess, and occur chiefly in the forms in which the spleen is the seat of overgrowth of pulp, as in splenic leucocythæmia.

The *Skin* may be invaded secondarily by the extension of growth from a lymphatic gland which has perforated the capsule. The papillæ of the cutis become infiltrated, the epidermis undergoes atrophy, and the glands are destroyed.

In rare cases subcutaneous nodules are observed. In one case such growths existed in front of the sternum. In other cases lymphoid tumours have been found in the substance of the cutis, the structure of which has been studied by Ranvier and Greenfield.[5] They consist of the same small cells and reticulate stroma, infiltrating the corium and the subcutaneous tissue and causing atrophy of the fat cells.

SYMPTOMS.—The two chief elements in Hodgkin's disease, the glandular enlargement, and the state of the blood, cause the most important symptoms of the disease. Others result from the generalised changes in important organs. It has been seen that the pathological changes vary much in different cases; they are attended by corresponding variations in the symptoms.

Earliest Symptoms.—Enlargement of the superficial glands is present, at some period, in almost all cases, and, from its obtrusive character, is often noticed early, having been the first evidence of the affection in two-thirds of the cases, 52 out of 78, in which the early symptoms were carefully noted. In two-fifths of the cases

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[1] Murchison; Schultz, Case 3.

[2] Murchison; Schultz, Case 1.

[3] Path. Trans. vol. xxi. 1870, p. 372.

[4] Mosler, Virchow's Archiv, vol. lvi. 1872, p. 14.

[5] Path. Trans. vol. xxvii. 1876, p. 278.

(36 out of 78) the first glands to enlarge were those of the neck. In some of these cases the glands were situated in the posterior triangle; in others, below the jaw. Enlargement of the glands on one side may precede for a long time the affection of those on the opposite side, especially when the glands to enlarge are those in the posterior triangle. The affection of the sub-maxillary glands usually soon spreads to the opposite side. In a much smaller number of cases, about one-eleventh (7 in 78), the inguinal glands were the first to enlarge. Less frequent still is an initial enlargement of the axillary glands. The glands first affected may remain enlarged for a long time before others are involved. In one case, recorded by Boorgard, enlargement of the tonsils was the first symptom of a glandular affection ultimately general.

When the deeper glands of the somatic cavities are those most diseased, the indirect symptoms which their enlargement causes may precede the affection of the superficial glands. Pain in the chest and cough may be the earliest symptoms in cases of enlargement of the mediastinal glands; pain in the abdomen, when the retro-peritoneal glands are the first affected; pain in the thigh or sciatic nerve, or œdema of one leg, when the pelvic and iliac glands are earliest enlarged.

In another group of cases the first symptom depends, not on the glandular enlargement, but on the accompanying blood state; and pallor, weakness, and emaciation first attract attention. In women these are often accompanied with arrest of menstruation. Dyspnœa is a constant attendant on the anæmia, and cough is often associated with it. In one or two cases epistaxis was the earliest symptom, and in two stomatitis. In a few cases slight general œdema, without kidney disease, was the earliest evidence of the affection; and in three cases on record the disease declared itself by irregular febrile attacks, preceding any other symptoms. In some cases the onset is accompanied by a febrile attack resembling typhoid, but of longer duration. A remarkable case of this character is recorded by Bohn, in which, after fever with splenic enlargement had existed for a fortnight, an attack of suppurating tonsillitis appeared to be the exciting cause of enlargement of the glands at first under the jaw, followed in the course of a few weeks by that of all the glands of the body.[1] These constitutional symptoms frequently accompany an initial glandular enlargement, but they alone ushered in the malady in about one-sixth of the cases in which the onset was carefully noted.

Glands.—The superficial glands when enlarged are usually firm and smooth. At first, and for a long time, they are separate, movable on one another beneath the integument; and this limited mobility is a peculiar feature, and has been thought to be characteristic. In many cases ultimately the mobility ceases, the glands becoming adherent to one another by peri-adenitis, or, more commonly, in consequence of their union by invasive growth. They then constitute an irregular lobulated tumour of considerable size, which may, by the pressure it exerts, cause very troublesome local symptoms.

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[1] Deut. Arch. für Klin. Med. 1869, p. 429.

The glands are usually firm, especially when of small size, and their growth of slow progress. Occasionally it is rapid, and the glands are much softer, and sometimes semi-fluctuating. When this is the case, they may contain such an abundant quantity of lymph that, a puncture being made, there may be a constant flow of lymph from the opening.[1]

The enlargement of the glands is usually attended with no pain, except such as results from the pressure they exert. Even in rapid cases the growths are usually painless. Sometimes slight pain in the swellings has been complained of. Occasionally when there is sudden swelling of the glands from time to time, the rapid enlargement may be attended with considerable pain.

The enlarged glands may continue to increase to the last. Frequently, however, an arrest of growth, and even a remarkable diminution in the size of the glands, may occur before death. It may be so considerable that the enlargement apparently subsides.[2] In the remarkable case recorded by Bohn early splenic enlargement occurred, and most of the superficial glands were considerably enlarged for about six months, but subsided before death, and, post-mortem, only the retro-peritoneal glands were enlarged, the spleen being simply hypertrophied.

The enlargement of the cervical glands generally begins in the posterior triangle or in the sub-maxillary group. The size attained by each group is often considerable, and the appearance of the patient may be strangely altered in consequence. The sub-maxillary glands may encircle the neck below the jaw, and the lower cervical glands may obliterate the neck and form a mass on which the head seems to rest as on the trunk. The enlargement may extend up to the mastoid process and parotid gland, and at the back of the neck the sub-occipital glands are also commonly enlarged. When the growth invades adjacent structures the substance of the thyroid may be involved. The more deeply-seated glands also enlarge and cause pressure effects, some of which have been mentioned in the section on Pathological Anatomy.

The pressure on the carotid arteries may so lessen the supply of blood to the brain as to cause cerebral anæmia. That on the veins may lead to the symptoms of passive cerebral congestion. Grave cerebral symptoms, coma, convulsions, &c., occur in some cases, and may be in part due to this pressure. The pneumogastric nerve is surrounded and compressed by the glandular growths, and sometimes this may be a cause of the cardiac irregularity and failure observed. The glandular growths may extend into the pharynx and fauces, and interfere with deglutition. Deafness is a very frequent symptom, and sometimes occurs early; it seems to be due to the pressure of the cervical and pharyngeal growths. The enlargement of the sub-maxillary glands may obstruct the movement of the lower jaw, and so interfere with mastication. The lateral displacement of the larynx may be such that, on laryngoscopic examination, one hyoid fossa may be invisible, while the other is wide open.

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[1] Bonfils, Recueil des Travaux de la Soc. Méd. d'Observ. 1857–8, t. i. p. 157.

[2] Bohn, loc. cit.; Caton, British Med. Journ. August 20, 1870.

The trachea is frequently compressed by the glands, sometimes so as to lead to very serious interference with breathing, and even to death by suffocation. When the compressed portion is just below the larynx, tracheotomy may be necessary and practicable, although most difficult. Trousseau gives a graphic description of the difficulties of the operation of opening the trachea, surrounded as it is by masses of enlarged glands and displaced vessels.

The œsophagus may be compressed by the glands in the neck or the posterior mediastinum, and dysphagia result. Sometimes there may be complete obstruction, and death may occur in consequence of inability to take food.

The enlargement of the thoracic glands may give rise to the signs of intra-thoracic growth, described in the article on that subject—dulness behind the sternum, and sometimes bulging of the chest-wall, displacement of organs, and various other pressure signs. Among the symptoms thus produced, dyspnœa and cough are frequent, especially from the enlargement of the bronchial and cardiac glands. The difficulty in breathing is spasmodic, and often produced in part by pressure on the recurrent laryngeal. Spasmodic cough may be also troublesome. Pressure on the large vessels is not common, but now and then is caused by a glandular growth in the anterior or posterior mediastinum. The aorta rarely suffers, but the superior vena cava is occasionally compressed, and all the signs of obstruction to the return of blood from the head and arms, enlargement of veins and œdema, may result.

The axillary glands, when large, interfere with the movement of the arm, and may exert pressure on vessels and nerves, and cause so much pain in the arm as to make their removal a matter of necessity for the relief of the patient. Enlargement of the epitrochlear glands may press upon the ulnar nerve and occasion severe pain along its course. The inguinal glands often compress the femoral vein, causing great œdema of the limb, and sometimes leading to thrombosis.

The symptoms which result from the enlargement of the abdominal glands are various. When the glands attain a considerable size, they may be commonly felt through the walls of the abdomen. In consequence of the lax parietes, pressure effects are less frequent than in the case of the thoracic growths. The retro-peritoneal glands, by pressing on the inferior vena cava, may cause or increase the œdema of the legs. When large, they may, by pressure on the stomach, lead to vomiting. They rarely interfere with the lumbar nerves, although the pelvic glands may compress the branches of the sacral plexus, and cause sciatic pain. The solar plexus may be surrounded and enclosed in a mass of glands, and then symptoms of Addison's disease may be present (p. 316). The portal glands may obstruct both the bile duct and the portal vein, causing persistent jaundice and ascites.

Spleen.—The enlargement is rarely great and does not often cause early symptoms. In this respect lymphadenosis presents a remarkable contrast to splenic leucocythæmia, in which early and obtrusive splenic symptoms occur. Only in rare cases are there pains in the left hypochondrium, or any sense of abdominal fulness from the splenic tumour. Now and then the pains are considerable, and may radiate to the back, and the organ may be tender on pressure. Usually the enlargement is sufficient to enable the spleen to be readily felt, and in some cases the tumour which it forms may be so considerable as to extend to the middle line, and then there may be symptoms due to the bulk of the tumour, and which are described elsewhere. When the nodules of growth are large, its surface may be perceptibly irregular. The spleen, like the glands, may lessen in size before death.

Liver.—The disseminated growths in the liver, if inconsiderable, cause no symptoms; when extensive, they may give rise to perceptible enlargement of the organ, rarely great, and commonly uniform, the growths not being so large as to cause any irregularity in its shape. They do not interfere noticeably with its functions. Even the infiltrating growth in the portal canals does not usually produce symptoms, probably because it rarely undergoes a fibroid change and causes little pressure. Jaundice, when present, is generally due to pressure of enlarged glands on the bile duct. Ascites is frequent, but is commonly to be ascribed to a similar cause, or is part of general anasarca. The dilatation of the veins of the liver, which is occasionally observed, no doubt assists in the production of portal congestion. The slight destruction of the liver-cells by the growth which infiltrates the acini, causes no recognisable symptoms—if any are produced, they are lost in those of the cachexia which is always present.

Kidneys.—The function of the kidneys, in most cases of lymphadenosis, is not materially deranged. In some cases the urine is dark and scanty. Few careful observations on its composition have been made. Dr. Wickham Legg found in one case a trace of indican reaction.

The disseminated growths in the organ may give rise to no symptoms, even when they are numerous. Sometimes, however, a trace of albumen is found in the urine, and now and then there are lymphoid cells resembling those of the growths in the kidney. Although the amount of albumen from the scattered growths is rarely considerable, albuminuria is not unfrequent from parenchymatous changes in the organ, but these must be regarded as complications.

Sexual Organs.—No symptoms can be referred to the rare growths in the sexual organs, ovaries and testicles, excepting the enlargement of the latter, which is perceptible on examination. When degeneration occurs in the new growth and the gland shrinks in consequence, the testicle may lessen in size to very small dimensions, as in a case under my own observation. No facts are on record concerning sexual power in the disease. It is probably only interfered with when the testicle becomes the seat of growth. In women amenorrhœa is common, and is often an early symptom. There are, however, many cases in which women have conceived and borne children after the affection has made some progress.

Alimentary Canal.—The development of lymphoid tissue in the various parts of the alimentary canal frequently gives rise to symptoms.

The stomatitis which has been already mentioned, is in some cases due to the development of lymphoid growths in the gums, in others it is the result of the blood-cachexia. It may occur independently of any excess of white corpuscles in the blood. The gums may be swollen and pale from lymphoid growth, or they may be spongy and even gangrenous. Vesicles may form on the swollen gums, and the secretion from them, usually fœtid, may be acid in reaction. Hæmorrhages may occur beneath the mucous membrane.

No symptoms attend the enlargement of the follicles of the tongue, but that of the tonsils causes some difficulty in swallowing, rarely considerable unless pharyngeal growths co-exist. It is probably by the interference of these growths with the function of the Eustachian tube, that the deafness so frequently noted is produced. The pharyngeal growths may cause troublesome dysphagia, both directly by their size, and indirectly by infiltrating the pharyngeal muscles, causing degeneration of the fibres, and consequently a muscular paralysis of the pharynx. In rare cases these growths may lead to complete obstruction, and consequently death by starvation.

The trifling œsophageal growths which have been noted in a few cases lead apparently to no symptoms. Œsophageal obstruction is always due to pressure of glands outside the tube.

The gastric function may be deranged in consequence of the local growths in the mucous membrane of the stomach, pressure from adjacent glands, or from the effect of the blood state. Loss of appetite, dyspepsia, and vomiting, are the chief symptoms of gastric growths. The vomiting is sometimes troublesome and persistent. When the growths, ulcerate the symptoms of gastric ulcer may be present, pain, tenderness, and hæmatemesis. The pain is less than that of simple ulcer. These symptoms may themselves lead to death. Very troublesome symptoms of dyspepsia with vomiting may result from the pressure on the stomach of the adjacent glands. In the commencement of the disease the appetite is usually undisturbed, although excessive hunger has been observed in young persons. In the later cachectic stage, anorexia is usually a prominent symptom.

The lymphoid growths in the intestines may be unattended by any indication of functional disturbance. In other cases there is long-continued and obstinate diarrhœa, sometimes accompanied with hæmorrhage. Diarrhœa may be troublesome, when there are no intestinal growths. It is common towards the end, and occasionally is marked as an early symptom. Even when the lymphoid growth is very extensive, the calibre of the bowel not being encroached on, no symptoms of obstruction result. Pressure on the intestines by a mass of enlarged glands outside it may lead to troublesome constipation, although rarely to actual obstruction. It is said, by Müller, that hæmorrhage from the rectum may result from the same cause, external pressure.

Respiratory System.—Disturbances of the respiratory organs are very frequent. Dyspnœa, due to the blood state, and felt chiefly on exertion, is an almost constant symptom, and is present in some cases throughout the disease. The respiration is often frequent—24 to 36 per minute. Considerable and permanent dyspnœa is usually due to pressure on the trachea or bronchi, or to interference with the pneumogastric nerve or its recurrent branch. As in other cachectic states, bronchial catarrh is common, and increases the dyspnœa.

The growths in the lungs are not usually so extensive as to give rise to physical signs. When of rapid formation, and attended with vascular disturbance in their vicinity, râles may be heard in places through the lungs. In the rare cases in which softening occurs there may be the physical signs of excavation.

Pleural effusion is common, and may result from pressure on the azygos or bronchial veins. When due to actual pleurisy, it must be regarded rather as a complication than as one of the symptoms of the disease.

Circulatory System.—The heart's action is not usually interfered with by the pressure to which it is occasionally subjected; nor have any special symptoms been noted in the cases in which growths have been found post-mortem in the walls of the heart. Fatty degeneration, due to the anæmia, may lead to the signs of cardiac feebleness and even to occasional attacks of syncope. The heart's action is quickened in proportion to the amount of febrile disturbance which may be present. It may also be disturbed, as already mentioned, by pressure on the pneumogastric.

Blood.—The anæmia is one of the most conspicuous features of the disease. It may be the earliest symptom, or the glandular enlargement may occur and advance before any signs of deficient blood-formation present themselves. We are able to estimate the amount and progress of the anæmia by means of the hæmacytometer, and may thus probably demonstrate its presence in all cases in which other symptoms of the disease are present. In patients with this disease in whom the face was well coloured, I have found the corpuscles reduced to sixty per cent. of the normal. In some cases there are many small red corpuscles.

In the majority of cases there is no excess of white corpuscles in the blood. In a minority of cases there is an excess, slight or considerable. Out of sixty-four cases in which the blood was examined by the microscope, there was no excess of white corpuscles in thirty-nine. In the twenty-five cases there was an excess, which in nineteen was moderate, in three was slight. In three others there was no excess of leucocytes during the early period of the case, but a slight excess was present during its later stages. The white corpuscles are as a rule small, corresponding with the description originally given by Virchow of their appearance in "lymphatic leukæmia." To this rule, exceptions are, however, occasionally met with. The cells are in many cases of different sizes; and when the excess is moderate, to ten or twenty times the average, the corpuscles may be of normal size. In most cases in which the increase was considerable it has been vaguely described, but in some it has been definitely stated. It very rarely, however, reaches a degree comparable to that which is frequent in splenic leucocythæmia.[1] In many of these cases the spleen was enlarged. To this point further reference is made in the section on Pathology.

The blood when drawn is strikingly pale, and has been compared to diluted claret. Except in the rare cases in which the leucocytes are in great excess, it has not the opaque appearance of leucocythæmia. After death it coagulates slowly and imperfectly.

The anæmia causes dyspnœa on exertion, due to the diminished number of oxygen-carriers in the blood. Langour and muscular weakness are marked. Other disturbances of the circulation also result from the anæmia. Œdema of the feet is common, and there may even be general anasarca from the same cause. Subcutaneous extravasations in the legs may occur, and hæmorrhages, especially from the nose, are frequent, but far less so in this disease than in splenic leucocythæmia. The occurrence of hæmorrhage may be independent of any increase in the white corpuscles of the blood.

The anæmia, and the emaciation which is probably to a large extent dependent on it, and the accompanying weakness and malaise, constitute what has been termed the "cachexia" of the disease. It is only absent in cases which terminate at a very early period from some accidental cause.

Nervous System.—The ill-nourished brain may present various evidences of weakness; depression of spirits, langour, and, in women, hysterical phenomena. The pupils may be unequal, from pressure on the sympathetic in the neck. In a few cases very remarkable nervous symptoms occur in the course of the disease, especially towards the close. These are delirium, coma, and epileptiform convulsions.[2] When death has occurred, no pathological condition has been discovered to which the symptoms could be referred. They occur independently of increase in the white corpuscles.

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[1] Of the cases of primary gland disease in which the spleen was not enlarged, and a considerable excess of leucocytes was present in the blood, one of the most extreme examples is that recorded by Virchow as "lymphatic leukæmia" in which the blood in the heart was thought to be pus. Other cases with a spleen of normal size are described by Mulder, in which greenish white clots, post-mortem, consisted almost entirely of white cells, by Dr. Strangeways Hounsell, in which during life there was one white to fifteen red, and the spleen, post-mortem, weighed only 3½ ozs., and by Dr. Ward in which 200 white corpuscles were present in each field of the microscope. Cases with a "great increase" observed during life, and normal spleen, have been recorded by Kottmann, Hughes Bennett, and McCall Anderson. In a case of extreme gland growth, recorded by Vigier, in a man aged 53, with a spleen six inches long, the proportion during life was one white to four or five red. With overgrowth of splenic pulp, and no conspicuous growths, cases with one white to four red have been recorded by Cayrium and by myself, and cases of "considerable increase" by several observers. In a case of typical Hodgkin's disease, in which the spleen contained conspicuous growths, a proportion one white to three or four red was found during life by Ollivier and Ranvier, and in another "extreme excess," post-mortem, by Potain and Chaillou. Cases of slighter but considerable excess have been recorded by Mosler, Pouffard, Schmuziger, and others.

[2] Southey, loc. cit.

Skin.—Pallor of surface is almost invariable when the disease is advanced, and it sometimes exists from the first. There is a corresponding pallor of the mucous membrane. There is often anasarca, and then the appearance may resemble that of Bright's disease. The skin may be dry during the febrile paroxysms, but in many cases it is moist, and in some cases there is copious perspiration. The occasional occurrence of bronzing of the skin has been already mentioned. Certain skin diseases are not uncommon during the cachectic period, especially a papular rash upon the backs of the fore-arms and hands, furuncles, and pemphigus.

Temperature.—Fever, occasional or constant, is a frequent but not invariable symptom of the disease. It is present in two-thirds of the cases. Out of forty cases in which the presence or absence of pyrexia was carefully noted, it was absent, or present only during some final intercurrent inflammation, in thirteen cases, while twenty-seven cases presented elevation of temperature as part of the special disease. It is not easy to say, from examination of cases, on what the occurrence of pyrexia depends. It is not associated with any uniformity, either with the duration of the disease, or with its diffusion. Pyrexia is a little more frequent in acute than in chronic cases, but to this rule there are some striking exceptions. Age, however, has a distinct influence on the febrile character of the affection. In almost all cases of lymphadenosis under twenty years of age there is fever. Nine cases of the forty cases were under twenty years of age, and in eight there was pyrexia. Thirty-one cases were over twenty, and the temperature was found to be normal in twelve and raised in thirteen.

The pyrexia exists at the commencement of the disease, chiefly in cases which begin with general swelling of the glands. Its characters in different cases vary much, and its course is commonly irregular. Three types may, however, be recognised. In one the temperature is continuously raised, presenting very slight diurnal variations of a degree or a degree and a half. The highest temperature is sometimes in the morning, sometimes in the evening. Occasionally the temperature may descend to the normal, and rise again. The degree of elevation is usually from 100° to 103°. (Fig. 2.)

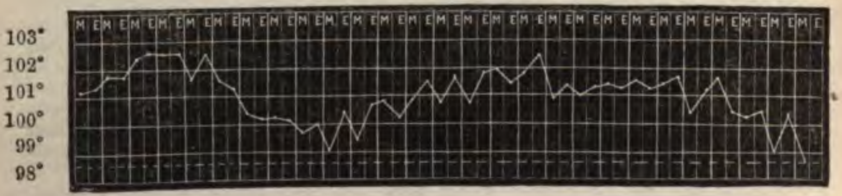

FIG. 2.—Temperature in Hodgkin's Disease: Continuous pyrexia.

A second type is characterised by periods of pyrexia, in which for several days a high temperature is maintained, the daily variation being slight. Alternating with these pyrexial periods are intervals of several days in which the temperature is normal or nearly so. The height attained by the fever may be considerable, sometimes reaching 105°, as in the case from which a chart is given. (Fig. 3.)

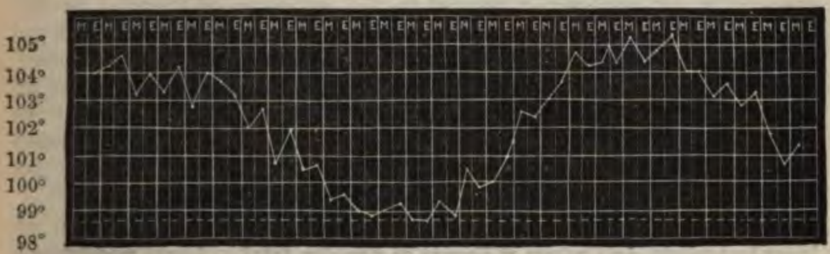

FIG. 3.—Temperature in Hodgkin's Disease: Alternating pyrexia.

A third type is characterised by morning remissions, the temperature being always higher in the evening than in the morning. The daily variations are from one to three degrees, the morning temperature being at or below 100°, sometimes normal, and the evening temperature being from 101°—103°, as in Fig. 4.

The same case may present at various times different types of pyrexia. In some the febrile periods are rare, in others pyrexia is the rule, and the intervals of normal temperature are the exception. Sometimes the pyrexial periods coincide with a rapid increase in the size of the glands.

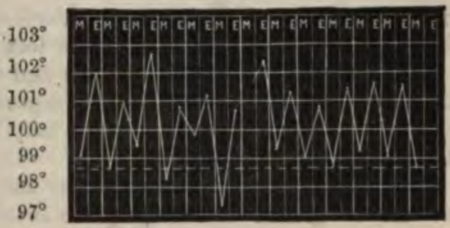

FIG. 4.—Temperature in Hodgkin's Disease: Remitting pyrexia.[1]

COMPLICATIONS.—The chief complications of lymphadenosis are (1) those which result from the pressure produced by the enlarged glands; (2) intercurrent affections of certain organs; (3) certain diseases resulting from the profound anæmia which is frequent; (4) some general diseases, perhaps indirectly connected with the glandular overgrowth, although not necessarily associated with it, of which the chief is tuberculosis.

(1.) *The Pressure Effects* of the glandular enlargements have been already discussed in considering the symptoms of the disease. Now and then their effects are so considerable, and run a course so independent as to deserve the name of "complications." Such are the thrombosis in the veins of the limbs, and the pleural and pericardial effusion, and the rare bronzing of the skin from disease of the solar plexus.

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[1] These charts are from cases in University College Hospital, under the care of Dr. Wilson Fox.

(2.) *Intercurrent Affections.*—The kidneys are occasionally the seat of parenchymatous degeneration, accompanied by albuminuria, and the other signs of Bright's disease. The occurrence of the change is apparently not necessarily related to the existence of growths in the organ, although the tendency of the scattered growths is to cause local degeneration. The kidney affection is usually insidious in onset, resembling rather a sub-acute than an acute change. Casts occur in the urine and sometimes present evidences of fatty degeneration. In one case which I have recorded the degeneration was possibly aided by the administration of phosphorus, but a similar degeneration has been met with in other cases in which no phosphorus was given.

Pneumonia is an occasional complication of the later stage of lymphadenosis, and when it occurs is usually fatal. Local pneumonia may accompany lymphoid growths, and in a few cases a caseating pneumonia has given rise to the slighter signs of phthisis. Tubercles have been found in the lungs in a few cases, but they occur only at the end of the disease, and give rise to no symptoms. Pleurisy is common towards the last, and the effusion is often great. It is apparently due in part to passive congestion, and in part to the cachectic state of the patient. Pericarditis has also been observed. Both are occasionally secondary to local growth.

Fatty degeneration of the heart and of the liver are not unfrequent and may give rise to their own symptoms.

The jaundice and ascites which may result from portal obstruction have been already described.

(3.) *Effects of the Anæmia.*—The anæmia and cachexia may lead to certain complications. Erysipelas sometimes occurs, especially in the parts the seat of œdema. Purpuric spots occur in the skin. Superficial abscesses sometimes form on the legs. Pemphigus has occurred in one or two cases, and in others there were troublesome boils; in one case at least a hundred in succession, followed by a papillary skin eruption.

(4.) *Certain general diseases* sometimes accompany lymphadenosis, and mixed affections result; one of these is tuberculosis. A more frequent but still rare accompaniment is scrofula. The characteristic of scrofulous affection is the tendency of the new formation to caseate, soften, and break down, and this tendency may be seen in either glands or spleen. The case described by Morgagni (note, p. 307) is an instance in which the distribution of lymphadenosis was combined with the anatomical characters of scrofula. In other cases some glands present the cheesy scrofulous aspect, while others have the characteristic appearance of lymphadenoma. In others, again, the glands have the hard glistening appearance of lymphadenoma, while the new growths in the spleen caseate, and soften.

COURSE AND DURATION.—The onset of the affection varies in different cases. In one group the general glandular enlargement is preceded for a long time, it may be for years, by local glandular swellings. In another the affection begins with a simultaneous enlargement of all, or nearly all, the glands in the body. Subsequently the evidences of impaired general health and nutrition are developed. Three stages have been proposed by Ollivier and Ranvier. The first is that of the early development of local gland-change, the second that of the generalisation of the affection, and the third that of the cachexia. It is to be noted, however, that the relative order differs in particular cases, and even the evidences of these as distinct stages cannot always be recognised. Occasionally the disease remains, for many years, limited to the glands first affected. In a case recorded by Jaccoud[1] an initial enlargement of glands in one axilla was followed almost immediately by those on the opposite axilla. Shortly afterwards, the enlargement of the latter lessened and disappeared, the glands first affected lessened in size and remained stationary for nine years; at the end of that time rapid generalisation occurred. I have a patient now under treatment in whom the first glands enlarged, in the right axilla, ten years ago, and were excised four years ago by Mr. Heath. Since then the glands in the right side of the neck have enlarged, but no others. The early latent period may, on the other hand, be entirely absent. The evidence of general anæmia may precede all the other symptoms of disease, or it may accompany the early local development of the affection. It is, however, progressive in character, and most pronounced towards the end.

The duration of lymphadenosis varies from three or four months to several years. The commencement of the affection can often be fixed with difficulty. On this account it is probable that some of the shorter cases are really of longer duration than they appear.

The duration of fifty fatal cases in which it could be fixed with some accuracy is given in the following table:—

Less than 1 year	in 18 cases.	Between 3 and 4 years in	6 cases.
Between 1 and 2 years in	15 ,,	,, 4 and 5 ,,	3 ,,
,, 2 and 3 ,,	6 ,,	Over 5 years	1 case.

The average duration of the whole fifty cases was nineteen months. Two-thirds of the cases terminated in less than two years. In a few instances the disease appeared to have lasted for more than four years, but these are cases in which a local glandular enlargement preceded for many years the general affection. If this period of slight local glandular enlargement were included in the duration of all cases in which it occurs, I believe the average duration would be much longer than is given above.

It is probable that this number of cases is too small to furnish any trustworthy evidence of the influence of age in the duration of the affection. As far as it goes it shows that the duration is nearly the same during the first half of life, and that in late life it becomes shorter. Thus in the following table the maximum, minimum, and average duration in months in each decade is given, and the number of cases on which the calculation is founded:—

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[1] Leçons de Clinique Médicale, p. 10.

Age at Commencement.	Cases.	Minimum.	Maximum.	Average.
1—10	9	4	36	19
10—20	9	3	54	18
20—30	9	6	42	18
30—40	11	4	48	22
40—50	2	18	60	—
50—60	7	8	24	14
60—70	3	6	8	7

The duration of lymphadenosis appears also to be little influenced by sex. The average duration of thirty-five male cases was just eighteen months, and that of eleven female cases nineteen months.

A few cases are on record[1] in which general swelling of glands and spleen, accompanied with a great excess of white corpuscles in the blood (in one case 1 white to 3 red), ran a rapid course, ending in death in a few weeks. They can hardly be classed with ordinary cases of Hodgkin's disease.

Termination.—Asthenia is the most common cause of death. The anæmia progresses, the patient becomes gradually weaker, more prostrate, anasarca occurs, the minor disturbances which attend a progressive cachexia increase, until at last the patient sinks, worn out. In a considerable number of cases death occurs in consequence of some accidental result of the morbid process. Of these the most common is asphyxia from pressure of the enlarged glands upon the trachea or bronchi, increased, in some instances, by laryngeal paralysis from pressure on the nerves. In eight recorded cases asphyxia was the cause of death. In a few cases the pressure upon the œsophagus has caused death by starvation. In one case, the instance communicated by Sir Robert Carswell to Dr. Hodgkin, the same result followed the occlusion of the pharynx by growths. Hæmorrhage from the mouth and nose was the cause of death in one case. Diarrhœa, although it often lessens the patient's strength, is recorded as the actual cause of death far less frequently than in splenic leucocythæmia. In a few cases death has occurred from cerebral disturbance, the nature of which is uncertain, manifested by coma, delirium, and convulsions, without discoverable pathological cause. Attention has been called to this circumstance by Dr. Southey, but it is certainly not so frequent as the number of instances met with by him would suggest.

In many cases death occurs from some intercurrent affection. Pneumonia is one of the complications most frequently fatal. Œdema of the lungs is common, and frequently assists other causes, especially pleural effusion, in bringing about the end. Pleural effusion is very common towards the last, but is not alone a frequent cause of death Rarer fatal complications are peritonitis, diphtheria, and kidney disease.

PATHOLOGY.—The changes in the lymphatic glands are allied to two morbid processes, inflammation and growth, so often, under various circumstances, combined. In some cases, the exciting causes of the disease appear to act by inducing a process of inflammatory irritation, and the alteration in the glands may be the same as attends such a process. More frequently, however, such exciting cause is not to be traced, and the structural change has the character of that independent and progressive tissue formation which we term a "growth."

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[1] Paterson, Edin. Med. Journ. 1870; Ponfick, Virchow's Arch. 1872, Bd. 56.

An attempt to assign the disease a position, according to the features of the growth which characterises it, is met at once by a new difficulty, resulting from the contrast which is presented by its clinical history and anatomical characters. Structurally, the growth is homoplastic, differing, it is true, from the normal structure of the glands, but differing sometimes little, and always by gradations, from the structure of glands which are enlarged under the influence of simple irritation, recent or remote.[1] Clinically, the disease has the character of malignancy, attaining a wider extension than any other morbid growth, not excluding tubercle itself, and attended by constitutional symptoms, as striking as those of cancer. This contrast, and the difficulties which it involves, were clearly recognised by Craigie, and we cannot wonder that he admitted that the word "cancer" accurately describes its clinical character, or that one of the latest writers on the disease finds in the term "desmoid carcinoma" its most accurate designation. The opinion which seems to harmonise best with the facts is that which has been advocated by Dr. Wilks, who assigns to the disease a position intermediate between cancer and tubercle, the change in the glands being similar to cancer in its clinical and naked-eye characters, and the dissemination of the growth, the affection of some of the internal organs, and the pyrexia of many cases, resembling the characters of tubercular disease.

The question whether the disease is primarily general or local has also been much discussed. Dr. Wilks pointed to the wide-spread distribution of the affection as evidence of a preceding blood disease. Wunderlich also urged that it must be regarded as a disease of constitutional origin, a general disturbance of the lymphatic system. The same view has been advocated by Müller and Schultz. On the other hand, Trousseau laid stress on the fact that the disease is, in some cases, distinctly started by a local irritation, and he believed that it is, in the first instance, a local affection, and that its subsequent development is a rapid generalisation. But this theory is not applicable to cases in which many groups of glands enlarge together without discoverable exciting cause. The characters of the disease certainly suggest that it is due, in all cases, to both constitutional and local causes, and that the extent of the two elements differs in different cases.

The general condition underlying the anatomical changes has been supposed to be an alteration of the blood, and the early anæmia and considerable pyrexia of some cases lend support to the view. But in most instances indications of a preceding blood-change are absent, and the general condition may be better conceived as a primary condition of the lymphatic tissues rather than of the blood. The assumption of such a predisposition is necessary to explain not only the cases which present a simultaneous affection of all the glands, but also the character of an initial local glandular enlargement, by which it persists in its special form. On the other hand, the hypothesis of an infective element in the primary local disease is hardly compatible with the phenomena of some of the cases for which it is especially needed, in which a local swelling exists for many years in a stationary state before any indications of generalisation present themselves.

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[1] Burdon-Sanderson, Path. Trans. 1870.

But the theory of infection cannot be summarily dismissed, because we must infer also the existence of a special predisposition to the disease, affecting both the glands first enlarged and those subsequently diseased. It is conceivable, and the phenomena of some cases suggest, that the two processes may both take part in the development of the disease. There may be an infection of predisposed tissues. Such double influence may have been at work in the remarkable case recorded by Bohn, in which a febrile attack with splenic enlargement preceded for a fortnight the glandular swelling, and the latter was apparently excited by a tonsillitis, the sub-maxillary glands being first affected, and afterwards, in the course of a few weeks, all the glands of the body.

The process of infection must be conceived as a contamination of the blood, since the generalisation is to glands and lymphatic structures out of the course of the lymph stream from the glands primarily affected. But the theory of infection, if accepted, is of limited application to the phenomena of generalisation. The localisation of the secondary growths is not that of most secondary tumours, and requires the assumption of a predisposition of the lymphatic tissues.

It is thus necessary, in order to explain the phenomena of Hodgkin's disease, to assume the existence of a general dyscrasia, affecting the lymphatic tissues, of different intensity and different operation in different cases, but existing in all. We must regard the affection as primarily constitutional, with local growths as the result of that general state, which are determined often by local causes. The local growth has received the name of lymphadenoma, and to the general disease, in these articles, the name lymphadenosis is applied. The closest alliance of the affection is thus with other diseases, especially tuberculosis, which are distinctly constitutional, and have, as their anatomical character, disseminated local growths, the occurrence of which is also distinctly influenced by local causes.

There are, however, certain forms of glandular growth allied to Hodgkin's disease which present important differences. A local growth may occur, lymphadenoma in structure, confined to a single group of glands, the other lymphatic structures of the body being free from disease. Similar growths may occur in the alimentary canal, and, rarely, elsewhere. In these cases the morbid tendency appears to be local, not general. Other forms occur (such as a visceral growth with nodules in the spleen) intermediate between these cases and those more general affections to which the term Hodgkin's disease is fairly applicable.

Other cases, again, differ by the opposite characteristic. In them there occurs sudden general glandular enlargement, commonly attended with an excess of white corpuscles in the blood, and so rapid and acute as to lead to death in a few weeks. Such are the cases recorded by Dr. Paterson, and already mentioned (p. 312). They had the aspect of intense glandular irritation, but without tendency to suppuration.

Some forms of scrofulous affection of the glands are also related to lymphadenosis. The characteristic of the scrofulous change in the glands is the low vitality of the tissue elements. In the early stage of enlargement the two conditions are very similar, but the elements of the scrofulous gland quickly die, undergoing fatty degeneration, and leading to general caseation or suppuration. There are cases, already mentioned, which present a mixed change, in which the characters of lymphadenosis and scrofula are blended, and some glands suppurate or caseate while others, with the spleen, present the changes characteristic of Hodgkin's disease, or in which the glands are enlarged as in Hodgkin's disease, and the spleen presents the cheesy softening nodules of scrofula.

The relation of lymphadenosis to splenic leucocythæmia must depend on the view which is to be taken of the latter disease. According to the general principle adopted in these articles, the existence of leucocythæmia furnishes no reason for separating certain cases of primary glandular enlargement from the rest. In the preceding article it has been held that splenic leucocythæmia depends essentially on the hypertrophy of the extra-lymphatic splenic pulp, and that the blood-state is conditioned by the imperfect development of the cells which are produced in the spleen, and which pass into the blood. We have in the splenic pulp a lymphatic tissue which is of, but not in, the lymphatic system, which possesses remarkable relations to the lymphatic system, of which the Malpighian corpuscles are part,[1] but which is so far outside the lymphatic system that it may be the seat of overgrowth without the occurrence of a corresponding overgrowth in any part of the lymphatic system, or even of detached lymphoid growths, without such growths elsewhere,[2] whereas the occurrence of growths in the Malpighian follicles is usually associated with the occurrence of growths elsewhere in the lymphatic system. An hyperplasia of the splenic pulp may, however, be conjoined in any degree with morbid changes, overgrowth, &c., in the lymphatic system, including the Malpighian follicles of the spleen, and hence arise the mixed forms of disease alluded to in the preceding article as "lymphadeno-splenic leucocythæmia."

The distinction between the cases in which the glandular swellings are hard and those in which they are soft was first made by Virchow, on the ground that the difference corresponds with a difference in the state of the blood, an excess of leucocytes being present in the blood in the former, and absent in the latter cases. He proposed to separate the soft cases as "lymphatic leucocythæmia," suggesting, however, that after all the distinction might be unessential. But many cases of the soft form without leucocytal excess in the blood are on record,[1] and although there are unquestionably some clinical differences between the two forms, neither their history nor their pathological anatomy justifies their separation as distinct affections. The two conditions may be conjoined, the superficial glands may be hard, and the deeper glands soft, or the same glands may be at first soft and ultimately hard.

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[1] It was the original idea of Malpighi that the follicles were the origin of the lymphatic vessels of the spleen, and Giesker also maintained that they were more closely allied to the lymphatic system than the splenic pulp. (Untersuchungen über der Milz, 1835, p. 154, quoted by Virchow, Ges. Abh. p. 188.)

[2] Pye-Smith's case, Path. Trans. vol. xxi. 1870, p. 390.

The anæmia commonly accompanies the general glandular enlargement: very rarely being noted first. It appears therefore to be a consequence of the gland disease. The opinion now generally held that the white corpuscles are the source of the red, and are themselves formed partly in the lymphatic structures, glands and splenic follicles, renders the blood-change in this disease intelligible. The change in these structures must, in the indurated form, arrest the formation of the cells, or their passage to the blood, and cause an anæmia, progressive as the gland disease progresses. The deficiency of red globules in these cases is some evidence that the corpuscles from the lymphatic glands (as well as those which are formed elsewhere) undergo transformation into red discs. If so, it is intelligible that when the new growth is such that the cells can pass into the blood (as in some of the soft forms) they may be so altered in their tendency of development that they may persist in the blood and give rise to an excess, more or less considerable, of white corpuscles. But an excess of leucocytes is not to be ascribed in all cases to the glandular enlargement. In most cases the spleen is also enlarged; in many it presents not merely the isolated growths, but an hyperplasia of the splenic pulp; in some this hyperplasia is the only change. But this is the alteration which is found in splenic leucocythæmia; and to it the excess of white corpuscles is commonly attributed. It is probably also a source of an excess of white corpuscles in many cases of Hodgkin's disease. It is so, almost certainly, in the mixed cases in which the spleen is very large, from hyperplasia of the pulp, and the excess of leucocytes in the blood is very great (1 to 3 or 4). It has been urged by Dr. Wilks that in all cases the excess is to be attributed to the splenic change. But there are cases on record in which a moderate and even considerable excess was associated with a normal spleen (see foot note on p. 335), and it seems reasonable to ascribe the excess in these cases to the passage of the altered gland-cells into the blood.[2] Sœmerring long ago showed that enlarged glands are permeable to quicksilver injected into the lymphatic vessels, and Schmuziger found that the lymph channels in the enlarged glands in leucocythæmia were also permeable.

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[1] Cases recorded by Moxon, Bonfils, Leudet, Hérard, and others. Hence the term "pseudo-leukæmia" has been restricted to these cases, and the hard form has been distinguished as "lympho-sarcoma," "desmoid carcinoma," &c.

[2] The remarkable case described by Lücke affords indirect evidence of the possibility of this origin of leucocythæmia. A large axillary lymphadenoma had ulcerated into a vein. Post-mortem, the spleen was normal, and there were no other glandular growths. During life the numbers of the red and white corpuscles in the blood were equal. The cells of the tumour precisely resembled the corpuscles in the blood. (Virchow's Arch. 1866, vol. xxxv. p. 524.)

It may be said generally that the generalisation of the new growths is throughout the lymphatic system, and that the tendency to their occurrence is apparently in proportion to the amount of lymphatic tissue normally existing in the part. This chief seat of the tissue is the glands and the splenic follicles, there the chief development of the disease takes place. The collections of lymphatic tissue in the alimentary canal are frequent seats of the secondary overgrowth, and the lymphoid growths in organs arise in interstitial spaces which are now known to be continuous with the lymphatic channels, and occupied by a retiform tissue. But in each of these situations growth may occur alone or out of all proportion to that elsewhere, causing the isolated or preponderant tumours of certain glands, or of the alimentary canal, already mentioned.

DIAGNOSIS.—The chief diseases from which lymphadenosis may be distinguished are—(1) local gland lymphomata, with or without secondary extension; (2) spleno-lymphatic leucocythæmia; (3) scrofulous enlargement of glands; (4) cancer.

A local gland lymphoma, single or spreading only to the glands next in the course of the lymphatics, is separable, as a local disease in its position and extension, from cases in which other growths exist in situations in which they can only arise in consequence of a blood infection or general predisposition. Where such wide generalisation has taken place, the cases are inseparable, by any anatomical feature, from the typical forms of lymphadenosis. Even the cases of simple local growth cannot be sharply separated from cases of Hodgkin's disease, since in the latter affection certain glands may precede and greatly preponderate over that of the rest.

Spleno-Lymphatic Leucocythæmia.—From a pure splenic leucocythæmia their distinction scarcely needs statement. The characteristic of the special form is the absence of any initial glandular enlargement. The cases of splenic leucocythæmia with a late affection of the glands are distinguished clinically by the same character. But there are the forms of mixed affection, already many times referred to, in which there is a simultaneous enlargement of glands and spleen, accompanied with a leucocythæmic state, and in which the form of splenic enlargement is simple hypertrophy. According to the view adopted in the present articles, these are cases in which the two forms of disease are conjoined. The distinction from simple lymphadenosis consists in the early and considerable enlargement of the spleen, and in the large size of the white corpuscles of the blood, and, after death, in the absence of isolated growths in the spleen.

Scrofula.—The anatomical difference of the glandular enlargement in scrofula from that in Hodgkin's disease has been already pointed out as consisting mainly in the tendency of the tissue elements to an early death. Clinically, they are distinguished by the following characters. In scrofula the enlargement is usually limited to one or more groups of glands which have been subjected to a local irritation. The glands affected enlarge rapidly and then remain stationary in size, undergoing softening, or rarely remaining firm. In lymphadenosis the disease may commence with the affection of one or a few glands, but the enlargement persists without diminution. Scrofulous enlargement of glands occurs chiefly in the young, that of lymphadenosis may occur at any age. Hence, although early age does not furnish any distinguishing character, adult age is in favour of lymphadenosis. In scrofula there are the well-known characteristics of skin, lips, &c., the tendency to low inflammation of mucous membranes, and to diseases of the bones and joints, all of which are absent in lymphadenosis. In lymphadenosis there is the long duration and commonly progressive course of the affection, sometimes better and sometimes worse, but continuing (except in the rare acute cases) for one or more years. The connective tissues adjacent to the glands are frequently and early affected in scrofulous enlargement, rarely in lymphadenoma, and then only late in the affection. Lastly, the tendency of the elements of the scrofulous glands to decay shows itself by the tendency to suppuration, which is wholly absent in pure lymphadenoma. Scrofulous glands do not, as a rule, attain the large size which lymphadenotic tumours often reach, but, as Virchow pointed out,[1] the distinction is not of universal application, since scrofulous gland tumours do occasionally reach a very large size. Now and then mixed forms of disease are met with in which one or two glands in a case of lymphadenosis suppurate, and others undergo caseation. The fact must be borne in mind and an opinion formed from the general character of the gland.

In the cases in which all the glandular structures of the system undergo a scrofulous overgrowth and degeneration, as in the case quoted from Morgagni (p. 306), a diagnosis during life is almost impossible, and can only be made by the occurrence of secondary softening in the glands. It must be remembered that in lymphadenoma the common change in glands is an induration, especially in the local cases in which the diagnosis from scrofula is chiefly needed; and any distinct diminution in the consistence of glands, especially if accompanied by a simultaneous diminution in size, suggests a process of degeneration, and therefore a scrofulous nature. As long as the change is limited to one group of glands, the diagnosis between scrofulous enlargement and commencing lymphadenosis is, it must be confessed, often difficult and sometimes impossible, and it is necessary to wait until the development of other characters decides the question.

Cancer of the lymphatic glands may be confounded with lymphadenoma, but chiefly with the forms which consist in the generalisation of a lymphadenoma which begins locally. The microscopical distinction of the two affections is complete. A scirrho-encephaloid, or an encephaloid cancer arising in the glands, has its distinctive structure in cells and stroma. In lymphadenoma, although the stroma may, in rare cases, have some resemblance to that of cancer, there are no cells of epithelial type. The clinical distinction between the two consists in the slow extension of cancer to neighbouring glands, and in the localisation of the later growths in organs rather than in glands, and the freedom of the spleen. On the other hand, in lymphadenosis, glands out of the lymph stream are early involved, and the spleen becomes enlarged with secondary growth.

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[1] Krankhaften Geschwülste, vol. ii. p. 728.

PROGNOSIS.—In all cases in which the affection is widely spread the prognosis is most grave. When the growths are also considerable in size a fatal termination is practically certain. But the duration of the disease is various, and the progress sometimes is very slow.

Sex affords no guide in prognosis. Nor does age give much information. It seems, however, that cases in children run a little less rapid course than cases in later life, and that age is therefore a somewhat unfavourable element in the prognosis. Previous health is also significant. A preceding cachectic state, from some independent cause, is of grave augury: such cases run a rapid course. Those, on the other hand, in which the disease commences in the course of fair health are much more chronic. Among the most rapid cases are some in which the disease was preceded by a profound depression of general health, the result of imperfect nourishment, or some lowering affection, of hæmorrhage, or of parturition, and some general cause, such as a severe exposure to cold, lighted up the affection.

Wunderlich thought that the cases in which the glandular swelling begins at many points run a less rapid course, and are more amenable to treatment than those in which a local enlargement of glands precedes the general affection. But the statement is of doubtful accuracy. Among the examples of each form are some of rapid and some of very slow course, and a comparison of a large number of cases shows that many of those which begin locally are very chronic.

The prognosis is in one respect rather better in the soft than in the hard glandular enlargements, because if, in the case of the former, the disease is influenced by remedial agents, the glands may recover functional power, while the hard glands are usually so much altered in structure that their return to a normal condition is almost impossible. But, on the other hand, if the treatment has no beneficial effect, the course of the softer form is the more rapid of the two. The occurrence of a slight excess of leucocytes in the blood has little influence on prognosis. Temperature is a symptom which, on the whole, indicates a rapid course. This is especially true of the continuous elevation and early pyrexia. The occasional irregular attacks of pyrexia have little prognostic value. Of other individual symptoms perhaps the gravest is the occurrence of œdema, which usually precedes, at no long interval, a fatal termination. The degree of anæmia, and especially its tendency to increase, affords important information, and by the numeration of the blood-corpuscles we are able to estimate its degree and variation in a more definite manner. A considerable, and especially an increasing, excess of leucocytes in the blood also renders the prognosis worse.

TREATMENT.—The local commencement of the glandular disease, suggesting the idea of a primary growth infecting the system, has led to the extirpation of the enlarged glands. In many cases in which the glands first enlarged have been removed, the further progress of the disease has been apparently uninfluenced. In all cases in which the affection of the glands was widely spread, even although elsewhere slight in degree, the removal of a group of glands chiefly enlarged, has not hindered the increase in size of the remainder. But in other cases, in which such indication of widely-spread disease was absent, an operation has been followed by a very marked improvement in the patient's condition, by a delay in the occurrence of the disease elsewhere so considerable that it has been reasonably ascribed to the extirpation, and in a few cases the disease has been apparently permanently arrested.

These results are in harmony with the pathological conclusions already suggested. The phenomena of the disease point, on the one hand, to the constitutional nature of the affection, and on the other, to the operation of local causes, determining the early and preponderant affection of certain glands; and they do not exclude the idea of a malign influence exercised by the earliest growth, which, for want of a better term, we call infective. It is evident, therefore, that in proportion as the constitutional condition is conspicuous in a case, the extirpation of the tumours may be expected to be without beneficial influence, and conversely, as the indications of a constitutional affection are inconspicuous, and the co-operation of local causes may be assumed as the most important immediate influence, benefit may be anticipated from the removal of the chief tumour.

Many recorded cases illustrate these conclusions. The removal of glands in the presence of indications of a general affection has been so unsuccessful as to lead many surgeons to abandon the idea of operative interference in any case, especially since a tendency to general glandular disease may exist and be latent at the time of the operation.

On the other hand, some favourable cases have been published, especially by M. Verneuil, of Paris.[1] He removed from the neck of a man a glandular tumour, which extended from the skull to the sternum and threatened suffocation by pressure on the trachea. Seven years afterwards, the man continued perfectly well. In 1866 he excised a mass of glands the size of a child's head, and of two years' duration, from the axilla of a man aged fifty-five. A small gland in the neck increased in size after the operation, and when it reached the size of a turkey's egg was removed, being separated with great difficulty from the carotid artery and jugular vein. During the next few years one or two small glands enlarged and suppurated (arsenic being given internally). Six years after the first operation there were one or two small stationary glands in the groins and neck, but the patient was otherwise perfectly well, when he died of acute double pneumonia.[2] In another case an axillary tumour, with part of the axillary vein, was removed. A year later a gland enlarged in the neck, and afterwards others elsewhere, and the disease ultimately became general, and the patient died, having, however, had six years of comparative freedom.[1]

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[1] Bull. de la Soc. de Chirurgie, 1872, p. 396.

[2] Bergeron, Sur les tumeurs ganglionnaires du cou, Thèse de Paris, p. 116.

In the case mentioned above (p. 339) Mr. Heath four years ago excised a mass of glands from the axilla, which had been slowly enlarging for six years. One small gland left in the axilla did not increase in size. Three years passed before there was any other evidence of the disease, and then all the glands on the same side of the neck began to enlarge, and are now slowly increasing.

In these cases the patient's condition was certainly improved, and the course of the disease was probably retarded by the operation.

From a comparison of the experience of Verneuil, Panas, and others, the following conclusions may be drawn regarding indication and contra-indication for excision.

The cases which are most favourable for an operation are those in which the disease is localised in a single region of the body, in which there are no enlargement of the glands elsewhere, no enlargement of the spleen, no pyrexia, and no considerable anæmia. It may be doubted whether the presence of one or two enlarged glands elsewhere, if not increasing rapidly in size, forbids an operation, provided no other contra-indications exist. Enlargement of the spleen certainly lessens much the chance of benefit, but if the organ is small, stationary, and the anæmia is not great, an operation may be undertaken, since cases with merely an enlarged spleen often remain stationary for a long time. The occurrence of pyrexia must be regarded as absolutely contra-indicating an operation, and so also must considerable anæmia. The degree of anæmia cannot always be inferred from the appearance of the patient, and it is desirable that accurate observations should be made in future cases. Probably, when the blood contains less than 60 per cent. of the normal proportion of corpuscles, extirpation should not be adopted. An excess of white corpuscles in the blood has been regarded as precluding any chance of success. It is doubtful whether this is a rule of absolute application. From the facts already considered, it seems probable that a slight excess of white corpuscles, say forty or fifty per field, is often due to a local growth, and it is very rarely that a larger excess is present in any case in which the question of operative interference arises.

In some cases an operation may be justifiable for the relief of symptoms, when there is no prospect of influencing the progress of the disease. The chief conditions in which, for this purpose, extirpation may be necessary are—(1) when death is threatened by the pressure of an accessible mass of glands on some important part; (2) when, by their pressure on nerves, &c., the tumours occasion severe pain or great inconvenience; (3) when a preponderating tumour threatens to soften and ulcerate, and kill the patient much sooner than he would probably die from the general disease. In all cases, and especially in the last case, the risk of the operation must of course be taken into very careful consideration.

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[1] Bergeron, Sur les tumeurs ganglionnaires du cou, Thèse de Paris, p. 116.

A mass of enlarged glands generally has deep connections, often with important structures, and their extirpation frequently is very difficult. But the difficulties are said to be often less than they appear, in consequence of the readiness with which the glands "shell out" without the use of the knife to divide their deeper attachments.

Verneuil, indeed, after having twice ruptured and tied the jugular vein, urges that neither exposure of the arteries nor ligature of large veins materially increases the risk of the operation. It must be remembered that the occurrence of generalisation is not always indicated by symptoms, so cases, apparently the most favourable, may turn out badly. Verneuil removed a large tumour from the groin of a patient who was apparently healthy, but died of pyæmia, and in the liver and visceral glands were new growths, which must have existed before the operation.

Other methods of local treatment of the superficial glandular swellings have been employed, and have appeared useful in some cases. Rubbing and shampooing the glands has been followed by distinct diminution in their size, and probably is more useful when the glands are soft than when they are hard. It may act by assisting the passage of their corpuscles into the lymphatic vessels. The alternate application of cold and heat to the glands by douches has been recommended. Cossy reported a case in which, by douches, "massage," and the administration of iron, the glands were reduced in size one-half. The application of ice has also been recommended, but has in no case affected more than a very trifling reduction in the size of the tumours. The same may be said of treatment by compression, carefully carried out by Billroth. Niemeyer, for the same object, employed and recommended repeated blistering, and the same treatment diminished considerably the size of a mediastinal growth in a case reported by Müller. Painting with iodine has also been very frequently employed, but with little benefit.

Galvano-puncture of the glands has been tried by Leyden,[1] Lücke,[2] and Billroth[3] without good result. Von Mosengeil[4] lessened tumours by causing sloughs by its means; but the operation is extremely painful, and often leads to troublesome sores.

Various injections into the substance of the glands have been tried, especially by Lücke, Czerny, and others,—iodine, iodide of potassium, nitrate of silver, chloride of gold, quinine, carbolic acid, gastric juice, chromic acid, and arsenic. Most of the injections excited inflammation, abscess, &c., but no true resorption. Injections of arsenic alone have not appeared to do good, but, combined with the internal administration of arsenic, they were thought by Winiwarter[5] to be useful. One drop of Fowler's solution was injected on each occasion. Considerable radiating pain was occasioned, not immediately, but a few hours afterwards. Winiwarter found, on examining glands which were excised a few days after an injection, that in the seat of the injection red corpuscles were effused, thrombosis had occurred in the vessels, and the area affected was sharply limited without adjacent inflammation. Occasionally, however, inflammation, and even suppuration, may occur. The latter is to be avoided, since an abscess is slow to heal and interferes with the treatment. Suppuration occurs in the skin more readily than in the glands, and hence it is important that the exterior of the cannula be perfectly clean, and free from any of the liquid to be injected.

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[1] Salkowski in Virch. Arch. Bd. lii. p. 58.

[2] Kottmann, Inaug. Diss.

[3] Billroth, Wien. Med. Wochenschrift, 1871, No. 44.

[4] Von Mosengeil, Arch für Klin. Chirurgie, Bd. xv. p. 146.

[5] Arch. für Klin. Chirurgie, 1875, vol. xviii. p. 98.

General medicated baths have been used. Trousseau thought that baths of corrosive sublimate, administered three times a week, were useful. Various natural baths on the Continent have also appeared to do good in some cases, especially Kreuznach. The sulphur baths of the Pyrenees have also been recommended.

Of internal remedies, iodine, from its supposed influence on scrofulous affections, has been largely used both in the form of tincture of iodine and the iodides of iron and of potassium. They have, however, done little good. Iodide of potassium was strongly recommended by Wunderlich on account of the recovery of one, not very conclusive, case. The glandular enlargements (inguinal and cervical) had existed for nine days only before the onset of an attack of diphtheria, during which the iodide was commenced. During the convalescence from the diphtheria the glands lessened in size, and in eighteen days they had disappeared. The same occurrence has been noticed after other febrile diseases in cases of even longer standing. In a case recorded by Mader, the use of iodide of potassium was accompanied by a considerable diminution in the size of the glands, but the patient died soon afterwards, and it is not uncommon for the glands to lessen in size before death.

Cod-liver oil has been given with advantage in some cases, and seems most useful when any indication of a scrofulous diathesis was present. Quinine in a few cases has appeared to do good. In the anomalous case recorded by Bohn (p. 330) it appeared to have a distinct influence in arresting the fever and leading to diminution in the size of the glands. In most other cases, it has been useless, even in large doses.

Arsenic has been given without success by several observers, but cases in which it did good have been recorded. Billroth relates a case of a woman, aged forty, with enlarged glands of ten months' duration, varying from the size of a hen's egg to that of the fist, in the neck, axilla, and groin, and the spleen twice the normal size. A fortnight after arsenic was commenced the glands were noticeably smaller and the diminution continued, so that in two months only one gland remained on each side of the neck the size of a filbert, and the remaining glands were still smaller. In other cases its action was less decided. Its use in increasing doses, up to forty drops daily, has been recommended, especially by Winiwarter. Increased pain in the tumours and general fever (the morning elevation slight or absent, the evening reaching 101°—103°), preceded or accompanied the diminution in the size of the glands, and signs of gastric disturbance, with increased emaciation, occurred even in the cases in which the result was best. These symptoms, apparently the result of the toxic action of the arsenic, are evidently not without danger, for one patient, in whom the glands lessened remarkably, died of marasmus, and another of erysipelas consequent on the excision of a small gland for diagnostic purposes. My own experience of arsenic has not been favourable. In several cases in which I have given it I have been unable to attribute any improvement to its use, although an influence on the glands was evidenced by the occurrence of considerable pain in them. In the recorded cases in which benefit accrued the diminution in the size of the glands was soon observed. In all it is necessary to omit arsenic for a day or two when toxic indications are noted.

Phosphorus has been of late given in this disease. It was first used for lymphadenoma by Verneuil,[1] from whose clinique several cases have been recorded, in which the tumours lessened remarkably in size. In one case I found that a remarkable diminution in the size of the glands accompanied its use, and an excess of leucocytes in the blood was reduced from 1 white to 4 red to 1 white to 100 red; but the patient died, the immediate cause of death being disease of the kidneys, to the occurrence of which the phosphorus had possibly contributed. Fatty degeneration was present in many of the glands, and new growths in the liver. This case, in conjunction with the known effect of phosphorus, suggests that it may do good by causing fatty degeneration of the glandular growths. The case suggests also, however, the difficulty of obtaining this result without producing a similar effect on organs of the body the integrity of which is necessary for life. But it would probably be wrong to assume that this is the only way in which phosphorus acts. In two other cases in which its effect on the glandular disease was inconsiderable, there was a very marked and steady increase in the corpuscular richness of the blood, in each amounting to 20 per cent. A very marked effect in the removal of anæmia has been observed by Dr. Broadbent. In several cases on record phosphorus has been given without any good result.

Mercury was recommended by Oppolzer, who saw temporary improvement from the use of the decoction of Zittmann, an aperient mercurial preparation. Birsch-Hirchfield has tried the internal administration of carbolic acid without success.

Iron for the anæmia is generally useless alone, but has sometimes appeared to do good in conjunction with other remedies. Careful diet, fresh air, and general tonics help the maintenance of the patient's strength, and sometimes appear to influence the disease. Verneuil mentions the case of a student with a very large gland tumour in the region of the vessels of the neck, who went to live by the sea. During the next three years the tumour gradually lessened till it was only two-thirds of the former size.

For the complications of the disease special treatment may be required, and occasionally surgical interference is necessary, especially in cases in which the enlarged glands compress the trachea above a point accessible for operation.

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[1] In 1873. Grocler, Thèse de Paris.

ADDISON'S DISEASE.

BY SAMUEL WILKS, M.D., F.R.S.

DEFINITION.—A disease characterized by a gradual loss of strength and general discoloration of the surface of the body in connection with a chronic inflammatory change in the supra-renal organs.

HISTORY.—In the year 1854 Dr. Addison published his treatise "on the local and constitutional effects of disease of the supra-renal capsules," but it was as far back as 1849 that he announced to a medical society [1] his discovery of the malady which is now generally known by his name. Since then much controversy has taken place both in this country and on the continent, with regard to the correctness of his statements, and for a long period an incredulity was clearly manifested in the writings of many eminent authorities as to the very existence of such a disease as Addison described. At the present time, however, it may be safely affirmed that the malady known as Morbus Addisonii has a good foundation in our nosology, and that all investigations have tended to corroborate the remarkable accuracy and truthfulness of the descriptions given in his original treatise. The only fact of importance which has been discovered since the publication of his memoir is the uniformity of the changes found in the supra-renal bodies after death, Addison not being himself aware that the disease in the capsules was of a special kind. As regards the symptoms. Addison's description is as full and accurate as any that has been subsequently given; but as to the exact nature of the complaint, and the relation of the events which characterize it, he professed no knowledge, and these have remained up to the present time in the same obscurity, Indeed, if we except the fact of a better acquaintance with the uniform character of the morbid changes in the supra-renal organs, our knowledge of the disease is much as Addison left it.

GENERAL DESCRIPTION OF THE DISEASE.—Before proceeding to any details of the symptoms, we think we cannot do better than place before the reader the following forcible and succinct account of the complaint contained in the original treatise of the author. He says, "The patient gradually becomes languid, weak, and indisposed either to bodily or mental exertion; appetite impaired or entirely lost; the white of the eyes becomes pearly, the pulse small and feeble, or perhaps soft and compressible; the body wastes without presenting the dry shrivelled skin and extreme emaciation usually attendant on protracted malignant disease; slight pain and uneasiness is from time to time referred to the region of the stomach, and there is occasional actual vomiting, which in one instance was both urgent and distressing, and it is by no means uncommon for the patient to manifest indications of disturbed cerebral circulation. Notwithstanding these unequivocal signs of feeble circulation, anæmia, and general prostration, neither the most diligent inquiry nor the most careful physical examination tends to throw the slightest gleam of light upon the precise nature of the patient's malady; nor do we succeed in fixing upon any special lesion as the cause of this gradual and extraordinary change." "With more or less of these symptoms we discover most remarkable, and, as far as I know, characteristic discoloration taking place in the skin, sufficiently marked indeed as generally to have attracted the attention of the patient himself or the patient's friends. The discoloration pervades the whole surface of the body, but is commonly most strongly manifested on the face, neck, superior extremities, penis, and scrotum, and in the flexures of the axilla and around the navel. It may be said to present a dingy or smoky appearance, or various shades of deep amber or chestnut brown, and in one instance the skin was so universally and so deeply darkened, that but for the features the patient might have been mistaken for a mulatto." "If I see a patient who presents this peculiar discoloration of the skin, and with this a certain train and combination of general symptoms, a pearly eye, a feeble pulse, a disposition to strongly-marked anæmia, and a few other symptoms less constant, I say this is a case in which you will find disorganization of the supra-renal capsules."

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[1] London Med. Gazette for 1849, p. 517.

It will be seen from this description that there are three characteristic features of the disease, and that all other symptoms are trivial, or arise immediately out of these three. They are the constitutional symptoms, the local effects on the skin, and the changes found after death in the capsules, or, to be more definite, a marked asthenia, a general pigmentation of the surface of the body, and a chronic inflammatory change in the supra-renal bodies. These are the three elements of the disease, and present such striking and distinguishing features, that if the first two are found to be present the third may be safely predicated. It will be convenient to consider each of these three divisions separately; and first of all to speak of the changes which take place in the capsule itself, for by so doing we have a basis of knowledge respecting the chronic nature and duration of the disease.

ANATOMICAL CHANGES IN THE SUPRA-RENAL ORGANS.—These organs in a well-marked case of Addison's Disease are found wholly destroyed, being converted into a substance which, from its outward appearance, usually passes by the name "scrofulous;" when cut through, the investing membrane is seen to contain a large mass of morbid material closely resembling a firm scrofulous gland, and exhibiting not a trace of the original glandular structure. The evidence of this material being scrofulous rests on no very good foundation, and therefore it might be regarded, provisionally at least, as a result of the ordinary changes which take place in inflammatory lymph. A very similar substance when found in the lung and other organs is usually regarded as one of the transitional stages of an inflammatory product. As regards the very earliest change in the capsules no case has yet been recorded where incipient disease has been found; nor is this likely to be met with, unless the victim of it were cut off by some acute malady. Up to the present time, whenever the organs have been found diseased the whole structure has been affected, and there has been every reason to believe that their destruction has been the cause of the patient's death. Although, therefore, it is unlikely that we shall meet with a case where the very earliest morbid alterations are apparent, yet, as Addison's Disease is one which may be protracted over many years, we have an opportunity of observing the changes in the capsules through several later stages. These stages may be conveniently divided into three. In the *first stage,* or the earliest in which the disease has been met with (but not inadequate to cause a fatal result), the organs have been found much increased in size, reaching to two or three times that of the natural dimensions of the capsules in a state of health. At this period it is rare to find a trace of the original structure remaining, but in its place there exists a firm solid substance which the older pathologists were accustomed to style lardaceous, not however intending to signify that peculiar matter known in more recent times as amyloid. It is an albuminoid material of a greyish white colour, homogeneous in appearance, firm when cut, and on section having a somewhat translucent character. It is the earliest known change which the capsule undergoes, and at this time nearly all the original structure has been destroyed, or if any be left it is recognised only by small dark spots which are the remnants of the cortical substance. The supra-renal organ has thus been converted into a morbid mass or lump of albuminous material, having very slight resemblance to its original form. Whether this morbid matter be the product of inflammation or not, the fact must be remarked that an inflammatory process has been associated with the disease, for the investing membrane of the supra-renal body has become changed into a thick tough capsule, and this is found firmly adherent either to the summit of the kidney, or, if the disease be on the right side, to the under surface of the liver.

At a later period, or in the *second stage,* this albuminoid material undergoes degenerative changes, commencing in isolated spots, so that it presents on the cut surface a number of yellow dots; these increase in size until the whole of the diseased organ is seen to have scattered through it a number of rounded yellow masses, about the size of peas, and resembling in outward appearance soft yellow tubercle. It is far more common to find the capsule in this condition than in the first stage just described—that is, the diseased organ, when removed from the body and a section made through it, is seen to be composed of two constituent parts—the homogeneous translucent greyish substance already described as the primary formation, and imbedded in it the softer or more friable yellow deposit.

At a still later period, and this for the sake of distinction may be called the *third stage*, the whole of the originally formed material may be changed into this yellow substance, and now it is that it presents the ordinary appearance of a mass of yellow tubercle. During this degenerative process the whole organ shrinks, and becomes reduced to about its original dimensions. After this, still further changes may ensue, as in similar deposits in other parts of the body, and thus a drying-up process may take place, and a cretaceous matter be formed. It is therefore not unusual to find, in the midst of the yellow material before spoken of, some deposits of a chalky substance, and if the shrinking still continues the whole mass of morbid material may become even smaller than the original organ. In many cases a still further change ensues by a process of softening, and the solid material becomes converted into a creamy or pus-like fluid, so that when the capsule is cut through this may escape just as in opening an abscess. Herein lies the explanation of those recorded cases of supposed Addison's Disease where it is stated that no capsules were discovered, but a little purulent matter occupied their place.

These are the successive changes which the supra-renal organs undergo in Addison's Disease, which may be styled the albuminoid stage or that of enlargement, the stage of cheesy degeneration, and the final stage when this is completed, ending in cretaceous deposit or liquefaction. It is rare to meet with the organ in the primary enlarged condition, and it is not usual to find the final stage of softening, but in the majority of instances the disease will be seen in the intermediate stage, where a number of yellow amorphous masses are found set in a translucent substance. It is this more usual condition which has suggested the idea of tubercle, a term which could not with any propriety be applied to the earlier or later stages of the disease. When examined by the microscope the albuminoid material is seen to consist of irregular-shaped cells, elongated nuclei and fibrillated structure holding them together. The yellow portions display shrivelled nuclei with fat granules and amorphous molecular matter. The original deposit consists of a low organized albuminous material disposed to fibrillate, and which readily undergoes a decay into the yellow amorphous substance.

SYMPTOMS.—As the disease in the capsules is passing through the successive stages just described, so the derangement of the health becomes more and more manifest, until the characteristic features of it are fully developed. It may be that the disease remains almost unrecognised until the period of completion, but generally it is evident that the patient is a sufferer from some hidden malady long before the diagnostic symptoms present themselves. For the sake of lucidity and of formal arrangement, however, we may consider that the symptoms slowly increase in severity as the successive changes in the capsules take place. Thus at the commencement the symptoms have appeared so insidiously that they are scarcely recognised by the medical attendant; who would perceive nothing more than a general derangement of health, with weakness and indisposition to bodily or mental exertion. At this time there would probably be loss of appetite or anorexia. In exceptional cases the gastric disturbance may be present from the very first, and thus actual vomiting. In some cases, especially if carefully looked for, some slight discoloration of the skin might be observed, but as a rule this is not one of the earlier symptoms. At a later period, or after some months as the disease advances, the failure of strength becomes more manifest, especially on attempts at walking or on any slight exertion. The disturbance of the gastro-intestinal canal is usually more marked, and sickness becomes one of the most troublesome symptoms. With this depression there may be giddiness, swimmings, and sometimes pain at the epigastrium or in the loins. The pulse is very feeble, and now the discoloration may have become marked, so that the whole body has assumed a dark hue. In what may be called the final stage corresponding to a complete destruction of the capsules, the discoloration is sometimes universal, and very striking to the most careless observer; the sickness or loathing of food is more marked; the patient may have become so weak from nervous and muscular debility as not to be able to remove from the recumbent posture, the pulse is thready or scarcely to be felt, and with this extreme depression there may be added some nervous symptoms, such as delirium or convulsions.

The Asthenia.—The most universal symptoms, and perhaps the most important, are those dependent on the asthenia, a term implying an utter sense of prostration or powerlessness, and in the present instance without any adequate cause to produce it. It is most important that this should not be forgotten, since an opinion has prevailed amongst many who have had no experience of the complaint, that the constitutional symptoms in Addison's Disease are characterized by anæmia and wasting. This is not the case, as neither of these conditions is present. It is true that the patient has a pearly eye, which is all the more marked from the contrasted colour of the darkened skin; but the lips and gums are not bloodless, nor do the muscles after death present that pale colour seen in anæmia. The ordinary symptoms of anæmia, such as throbbing headache, palpitation, and breathlessness, are not those which more immediately characterize the presence of Addison's Disease. The symptoms are rather those of asthenia or want of nerve-power, exemplified by the extreme feeling of prostration on the part of the patient, and made known to the medical observer by the remarkably thready and feeble pulse. No symptom is so striking as the fact of the patient sitting in his chair not seeming very ill, perhaps conversing with those around him, and yet having so small a pulse that it can with difficulty be felt; or at a later stage lying in bed talking, or even, as the writer has seen, singing in a low voice, and yet be absolutely pulseless at the wrist. In such a case the medical man is not so much reminded of the feebleness dependent on poorness or loss of blood as of the prostration which is witnessed in cases of cholera or peritonitis. As the patient is not anæmic, neither is he wasted. It is an error to describe the subjects of Addison's Disease as emaciated. The patient is reduced in bulk and weight, but his limbs are rounded, and after death a good layer of subintegumental fat is found. The body does not present that withered dry appearance so often seen in abdominal cancerous disease. Associated with the asthenic condition, and probably as a mere manifestation of it, some other symptoms may be noted. The most marked of these is the sickness or vomiting. Sickness indeed has been an almost universal symptom, and in some cases of so urgent and persistent a nature that the patient has succumbed to its effects at a very early period of the complaint; and if this has occurred before any signs of discoloration have become manifest, the case may have been regarded as one of cancer of the stomach; if diarrhœa has been associated with the sickness, the case may have been styled one of gastro-enteritis. Such instances, however, are quite exceptional. At the same time there may be more or less pain at the epigastrium, between the shoulders, or in the loins. Other symptoms indicative of a perverted state of the nervous system may be observed; but they are those which might be expected to attend so depressed a vital condition. Thus during the progress of the complaint there may be neuralgic pains in the limbs, and towards the close delirium or convulsions.

The Pigmentation of the Skin.—In a typical case the whole cutaneous surface of the body is discoloured, and the patient, if he have before an ordinary white skin, now assumes the appearance of a native of a hotter clime, so that he may be asked if he have any dark blood in his veins. The colour at the commencement of the complaint is brown, or olive-green brown, and becomes darker or blacker as the disease advances. The greenish tint or walnut-juice shade (as an artist who has taken many portraits of patients has styled it) gave rise at a very early period in the history of the disease to the expression "bronzed skin," as indicative of this remarkable change. Although the discoloration is more or less uniform over the whole body, some parts are more intensely affected than others; thus those regions which are darker in the black races are most liable to pigmentation in this disease, viz., the genital organs, the axillæ, the areolæ of the mammæ, &c. The discoloration is also observed to be more marked in those parts which are more exposed to the sun, as the face and backs of the hands, especially over the joints, also those parts on which any pressure may have been exerted, as around the body by a waistband, or around the leg by a garter.

In all these parts where the colour is darker it is gradually shaded off into that of the surrounding skin; in no case does it cease abruptly or is the pigmentation in circumscribed patches. To the inexperienced, such instances of discoloration in patches surrounded by pale skin are often regarded as cases of Addison's Disease; but they are purely local affections and examples of leucoderma. In Addison's Disease, on the other hand, the discoloration is so uniform that in more than one case the patient has been regarded as a mulatto or man of colour. A closer investigation, however, would show the distinguishing features between them, for in the Englishman the hair might probably be light, and his eyes of a blue or grey colour. In only one or two recorded instances has it been observed that the hair has become darkened. In all cases which the writer has examined the scalp has retained its natural hue, the nail has preserved its whiteness, and the soles of the feet and palms of the hands have remained colourless. In some cases the discoloration does not cease with the skin; but pigment may be discerned on the buccal mucous membrane; so that the gums, inside of the cheeks and of the lips, may be sometimes found to present a few black spots. In one or two cases there has been a black patch on the tongue. It has been stated, but without apparent sufficient foundation, that pigment has been met with on the mucous membrane of the bronchi, intestines, genital organs, and other viscera. It is certainly sometimes seen on the peritoneum, but as this is occasionally found in other cases than in those of Addison's Disease, it cannot be said that there is any connection between them. At present, therefore, it must be affirmed that, with the exception of an occasional pigmentation of the buccal mucous membrane, there is little evidence of discoloration of the internal parts of the body. On examination of the skin by the microscope the pigment is found to occupy the cells of the rete mucosum in the form of dark-brown granules, just as in the dark races of mankind; the section shows the pigment following the undulation of the papillæ where it is more thickly deposited; whilst the true cutis beneath remains healthy.

ORDER OF SYMPTOMS.—Addison asserted that the more advanced the change in the capsules the more discoloured will probably be the skin of the patient, and likewise the contrary, that where the first albuminoid stage has not been passed no alteration in the colour of the skin will be expected. After the publication of his memoir, Addison met with a recent case of this kind where no discoloration was present, and since this, some few other similar cases have been observed. These facts would tend to show that the pigmentation of the skin does not occur at a very early period of the complaint, but is one of the later symptoms, and that the constitutional asthenia is really the most important pathological feature, and may be the only symptom present when the patient falls a victim to the disease. It would appear from this that the subjects of Morbus Addisonii are sufferers from the various manifestations of debility long before any apparent change is observed on the skin. Probably this is the case, although it may be true that when the patient is first seen by his medical attendant some discoloration is observable, since his symptoms until that time are not sufficiently severe to compel him to seek advice. It must, however, be admitted on the other hand, that occasionally the altered colour of the skin has been accidentally observed by the friends at a time when the patient had not regarded himself as being out of health. From this fact, and the analogous one of the skin being sometimes only slightly discoloured in advanced cases, the above rule can only be considered as a general one and applicable to the majority of instances. The fact, however, must be remembered that in some remarkable and exceptional examples the disease has run a rapid course to its end, and before the development of any pigment in the skin. It is for this reason and that because nervous depression is the only symptom which is invariably present, that one French writer has suggested the name "Asthenie Surrénale" for Addison's Disease, instead of "Melanodermie Asthenique," a term previously proposed.

DURATION OF THE DISEASE.—In some cases the history of the illness has been as brief as six months; in others it has been prolonged over several years, making an average taken from a large number of cases of about one and a half years for the duration of the complaint. It must be remembered that in those cases which have been termed acute, on account of the short illness and the severe nature of the symptoms, the capsules have been wholly converted into a new material, and therefore it is reasonable to suppose that as soon as the disease is recognised and symptoms appear, that the supra-renal capsules are totally destroyed. It will be thus seen that the above calculation applies only to the duration of the symptoms, as we have no data by which to determine the time necessary for the deterioration of the capsules. It is probable that this is very lengthened, and judging from the cretaceous deposit it is fair to conjecture that the disease is extended over many years.

MODE OF DEATH.—Death is generally slow, and ensues from an aggravation of all the symptoms of asthenia which have so long existed. The patient has become weaker and weaker, the heart's action more feeble, so that for some days before death he may be absolutely pulseless, and life in the gentlest manner ebbs away. Sometimes, however, the patient, having been reduced to the lowest state of vitality, remains in this condition for weeks and months, and then suddenly expires. He may indeed die slowly and imperceptibly like a plant, or, his vitality being all but gone, he may continue some time like a flickering flame and then in a moment be gone. As before said, there may sometimes be associated with the final catastrophe delirium, slight convulsions, or coma.

Age and Sex.—Addison's Disease is met with at all ages of adult life and in both sexes, but more rarely in children and in very old people. By far the larger number of cases have occurred in men before the middle period of life.

Association with other Diseases.—In the majority of recorded cases of Morbus Addisonii, no other disease than that in the capsules has been found in the body, and thus by a fair inference it has been supposed that the symptoms and the death of the patient have been dependent upon or intimately connected with the destruction of these organs. In some cases there has been observed a vascularity or apparent hypertrophy of the glandular structure of the intestinal canal as in diseases where a long continued irritation or much gastro-intestinal disturbance has been present. Thus Brunner's glands in the duodenum, and the solitary glands in the ileum and colon, have been found more than usually prominent in Morbus Addisonii. In some cases tubercle has been met with in the lungs, and this fact taken in connection with the peculiar change found in the capsules has confirmed the opinion in the minds of some that the disease is really scrofula, but it must also be stated that the deposit in the lungs has generally been of the same kind as that in the capsules, whose true nature has been the subject of discussion. In the majority of instances, however, the lungs and all the other organs are healthy. Should the disease be of the nature suspected, we have the very exceptional fact of tubercle existing in a part of the body for many years, sufficient in importance to kill the patient, and yet not found in the lungs. This would almost constitute a reason in itself for not regarding the disease of the capsules as scrofulous. Another complication which has been met with in a few cases is a caries of the spine, and the seat of the disease has been adjacent to that in the capsules. In some of these instances the history of the spinal affection has dated back long anterior to that more immediately associated with the capsules, and therefore if the two diseases have been in any way connected, that of the spine must be regarded as the older. The connection may be looked upon as a mere coincidence, but there seems no pathological objection to the conjecture that the supra-renal organs may have become affected in consequence of the adjacent caries of the bones.

Pathology.—There is the very strongest evidence in favour of the opinion that the change found in the capsule is peculiar, uniform in character, and primary in its nature. We mean by this that it does not consist of various forms of disease, and is not a mere accidental part of a malady affecting the body generally, but that it is as much a primary and essential disease as a cirrhosis of the liver, or a granular degeneration of the kidney. It is unfortunately true that Addison, not content with placing on record his few genuine examples of the complaint, hazarded a conjecture that cancer or any other destructive agency might develop the same symptoms, but he gave no instance in corroboration of it, and no subsequent writer has published a single case where cancer, tubercle, or indeed any form of benignant or malignant growth has been productive of the genuine symptoms, nor indeed has any case been recorded where degenerative changes other than those previously described have given rise to the complaint. In all the cases where the symptoms have been well marked the change in the capsule has been of one kind and essentially primary. A moment's consideration would convince the pathologist that a complaint having uniform symptoms as pronounced as any disease in the nosology, would have almost of necessity a counterpart rather in those diseases which we style Morbus Brightii, cirrhosis of liver, or phthisis, than in those which admit of such general expressions as kidney disease, liver disease, or lung disease, which include cancer, tubercle, abscess, or indeed any morbid condition to which the organ might be liable. In studying the diseases of these organs we speak of those primary affections just named, and not of any adventitious affections which may attack them from without. So in Addison's Disease all pathological and clinical considerations would make us disclaim the notion that any accidental diseases of the supra-renal organs would produce so uniform a set of symptoms as are always attached to it. All such considerations are opposed to it, as for example its duration, which is very different from the rapid course of these secondary affections; a cancer for instance would endure infinitely too short a time for the production of a complaint which lasts for years. Then again, the sole seat of disease being in the capsules and affecting both simultaneously would be sufficient to show its essential primary nature, since diseases of this kind would necessarily attack both organs simultaneously, and lead to their destruction without evidencing themselves in other parts of the body. Then again, although one would not wish to introduce any *à priori* argument into a question of fact, it might be presumed that a chronic affection of the nature of Addison's Disease would necessarily be dependent (supposing it to be due to a change in the capsule) upon a change analogous to that found in other chronic diseases, that is, one due to inflammation and its results; using the term of course as loosely as is done when cirrhosis is styled a chronic inflammation of the liver, Bright's disease a chronic nephritis, or many forms of phthisis chronic inflammation of the lungs. As regards the latter, the material commonly found in the lungs as a result of inflammation closely resembles very often that met with in the supra-renal organs.

We might mention that in several articles to be found both in French and German works on the subject of Addison's Disease, the authors have collected some hundreds of cases where the supra-renal organs were diseased, also cases where the skin was discoloured, and where asthenic symptoms prevailed. On carefully perusing these the position we have taken is clearly proved; that the symptoms which Addison described can be associated only with one form of disease of the capsules, although this is not the conclusion of the authors themselves, who have also mistaken cases of leucoderma and other cutaneous affections for it. Martineau, in his memoir, after collecting a large number of cases, says, "il est évident que la maladie d'Addison ne pourrait être considerée comme entité pathologique." A careful perusal however of his own cases might have enabled him to arrive at an exactly opposite conclusion.

Believing, then, that the change in the capsule in Addison's Disease is uniform, the question arises as to its nature, and whether it should be styled scrofulous or inflammatory. As regards the former term its physical characters alone are not sufficient to identify it since no specific peculiarities of scrofulous matter have yet been announced. Then, again, unless it be asserted that scrofulous matter always originates in the degeneration of a prior albuminous product, another objection would arise, for such a degenerative process is the one which occurs in the disease of the supra-renal capsules. Again, it may be remembered that the capsule is met with in the last stage of softening or chalky degeneration when none of the ordinary appearances of scrofula can possibly be seen. We should consider also that if we regard scrofula as a constitutional affection we have here the case of two organs being selected for attack and remaining the sole receptacle for the deposit for many years without any part of the body being affected. It has, nevertheless, been asserted by some that Addison's Disease is a manifestation of the tuberculous cachexia, and that the change in the capsules is the mere outward evidence of its presence, and therefore the latter cannot be regarded as the cause of the fatal issue any more than the discovery of an enlarged spleen can be looked upon as the cause of death in intermittent fever. If this were true it would constitute a very remarkable fact in the history of scrofula. Under what appellation then shall the disease be known? We think provisionally it might be called inflammatory. An albuminous material, it may be remembered, is formed which subsequently degenerates. Exactly under what circumstances this arises cannot be declared, but as in the present era of medicine we are in the habit of speaking of such productions as results of inflammation we may well adopt the term here. And it would not be denied that such a process had been present when the investing membrane is found thickened and adherent to surrounding parts. On the supposition of the disease arising from a chronic inflammation it would not be unreasonable to conceive it having its origin in a blow on the back, a strain, or a previous caries of the adjacent vertebræ.

Etiology of Symptoms.—The symptoms described by Addison in connection with the disease of the supra-renal capsules were of so remarkable a kind that immediately on the publication of his treatise a fresh interest was at once excited in the minds of physiologists as to the use of these organs. Experiments were first instituted with a view to the corroboration or otherwise of the deduction which was supposed to flow from Addison's clinical observations as to the vital nature of the capsules, for it seemed probable that their removal in a living animal would produce the well-marked symptoms which Addison described. It is remarkable that the first experiments on animals did show (it was said) an exactly similar train of symptoms, and that when these organs were removed there followed an increasing weakness, impeded circulation, fall of temperature, failure of appetite, convulsions, and death, whence it was considered that these organs were indispensable to life. There was found also an increase of pigment in the blood, and it was supposed therefore that the capsules were vascular glands and carried on the metamorphosis of a substance destined for the formation of pigment. Other experiments however soon followed, and the truth of all these statements was denied. Indeed, all further physiological research has tended to throw no additional light on the function of the supra-renal capsules. It had been too readily assumed by many authors that Addison in his treatise had suggested the vital nature of the supra-renal organs, whereas his facts tend distinctly to prove the opposite—that persons may live for years when the capsules are wholly destroyed. At least, this is the only conclusion we can arrive at when we consider the length of time necessary for the production of the cretaceous deposit which is sometimes found within them, being guided moreover by the experience of the changes witnessed in the mesenteric and other glands. Subsequent experimenters, especially interested in the subject of pigmentation of the skin, renewed their examination of the blood in connection with the colouring matters of the urine, the bile, and that of melanosis, and they then compared these with the dark pigment found in the healthy capsules. There was however little more discovered than the fact published by Vulpian that the blood of the supra-renal veins gave a special reaction to various tests, and that a peculiar substance which was formed in the substance of the organ was poured into the blood and gave a bluish tint with perchloride of iron. The blood too has been carefully examined microscopically and been said to present certain peculiarities in the shape of the corpuscles, in their relative proportion, in the deficiency of fibrin, in excess of colouring matter, &c., but all these statements have been denied by other experimenters. Various theories in explanation of the pigmentation have been advanced, but they cannot be sustained, since most of them would apply to a general discoloration of all the tissues of the body, and could not account for a pigmentation which affects the skin only, exactly as is seen in the dark races of mankind. From a consideration of all that has been written on this subject we are in a position to state that the immediate cause of the discoloration must at present be regarded as unknown, and also that the connection between the discoloration and the change in the capsule is equally obscure, whether indeed it be due immediately to destruction of that organ, or whether it be only one feature of cachexia, as sometimes witnessed in a modified form in tuberculosis or in old age, but which in the disease under consideration is slowly brought about by the destruction of the capsules.

As regards the asthenia, one of the three great phenomena which characterize Addison's Disease, it is remarkable that most of the experimentalists alluded to were agreed that a very decided and sometimes fatal depression succeeded an injury or crushing of the supra-renal bodies, just as when the neighbouring sympathetic ganglia were similarly treated, and thus Addison's own views on the cause of the asthenia gained considerable support. Addison had at once seen that these organs could not be considered vital in the ordinary acceptation of the term, since they might be functionally absent during the lifetime of the patient, and thus he was induced to consider whether their contiguity to the great ganglionic centres and the implication of the nerves of the latter might not account for the remarkable depression which is the most universal symptom of the disease, and these were his words:—"We know that these organs are situated in the immediate vicinity, and in contact with the solar plexus, and the semi-lunar ganglia, and receive from them a large supply of nerves, and who could tell what influence the contact of these diseased organs might have on these great nerve centres, and what share that secondary effect might have on the general health, and in the production of the symptoms presented." Since Addison thus spoke, Brown-Séquard and Mattei have shown that by crushing the organ similar results followed as when the sympathetic was experimented upon. The latter writer says "in a rabbit I violently compressed both capsules at the same moment. The animal gave a sharp cry and fell into a state of prostration; respiration almost ceased, and in a few minutes it was dead." Now it may be stated as a matter of fact that, besides the implication of nerve branches in the semi-lunar ganglia, the capsules have sometimes been so enlarged as actually to involve the ganglia themselves in their morbid mass. Such explanation for the asthenia also draws Addison's Disease into closer relationship with such an affection as peritonitis and cholera, where from the extreme depression we are forced to consider that the abdominal sympathetic nerves are subjected to a pernicious influence. The objection to this interpretation of the asthenia is that it makes the symptoms quite independent of the destroyed function of the organ and suggests a mere accidental implication of the nerves. Also it must be stated that no morbid change in the semi-lunar ganglia has yet been discovered, although they have been carefully examined by the writer and other observers. The remarkable combination of asthenia and pigmentation in Addison's Disease has suggested to some physiologists the double function of the organ, and thus whilst some have been regarding them as nothing else than nervous ganglia, and others as little more than lymphatic glands, Kölliker had advanced an opinion that the central portion might be classed with the vascular glands, and engaged in the function of secretion, whilst owing to the great nervous richness of the medullary substance, the latter might be an apparatus forming part of the nervous system. Brown-Séquard has established after Ecker, Frey, and Kölliker, that the peculiar cells

EXOPHTHALMIC GOÎTRE.

By Hermann Beigel, M.D.

Synonyms.—Grave's Disease; Basedow's Disease; Struma Exopl
thalmica; Goître Exophthalmique; Tachycardia Strumosa (Lebert
Cardio-thyroid Exophthalmos (Walshe).

Under one of the above titles a group of symptoms has been d
scribed, of which the most constant are Exophthalmos, Goître, an
Palpitations. Some authors[1] are of opinion that Flajani[2] had som
notion of the connection existing between these symptoms, b
after a careful examination of the numerous cases of exophthalm
which this author published, I have not been able to find anythin
confirming this opinion. It is true he mentions several times th
occurrence of palpitation in cases of exophthalmos, but he is by n
means conscious that the concurrence of these phenomena—he do
not mention goître at all—constitutes a special disease. In anoth
place he relates three cases of goître occurring with palpitation i
men, and in all these he made a cure by mere local treatment of t
enlarged thyroid gland; in these cases, however, he does not mentio
the existence of exophthalmos, so that we may safely say that Flaja
was not aware of the disease at present known by the name of Exop
thalmic Goître.

It seems that Percy[3] was the first to pay attention to the enlarg
ment of the eyeball in connection with the other two symptoms, b
he also did not recognize the real relation between them.

Graves[4] was the first who distinctly described three cases of pa
pitations and goître, and, when speaking of the latter symptom, l
says that the enlargement of the thyroid "seems to be essentiall
different from goître in not attaining a size at all equal to that o
served in the latter disease. Indeed, the enlargement deserves rath
the name of hypertrophy, and is at once distinguishable from bronch
cele by its becoming stationary just at the period of its developmen
when the growth of the latter begins to be accelerated. In fac
although the tumour is very observable when the attention is direct
to it, yet it never amounts to actual deformity." We shall see whe

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[1] Dr. Paul, Berlin Klinisch. Wochenschrift, 1867, p. 227.

[2] Giuseppi Flajani, Collezione d'osservazioni e reflessioni di Chirurgie, vol. iii. p. 27
Roma, 1802.

[3] Caleb Hilliard Percy, M.D., A collection from the unpublished medical writings
London, 1825.

[4] Robert J. Graves, M.D., A System of Clinical Medicine. Dublin, 1843, p. 674.

we come to the symptoms of the disease, that Dr. Graves' observations were perfectly correct, and still hold good. Besides these three cases he describes one[1] in which all three symptoms, viz., exophthalmos, goître, and palpitations were very marked. The latter developed first and produced a pulse "never under 120, and often much higher;" after about a year exophthalmos was noticed, and "in a few months the action of the heart continued with increasing violence, a tumour of a horseshoe shape appeared in the front of the throat, and exactly in the situation of the thyroid gland."

At about the same time Marsh[2] of Dublin and Pauli[3] treated of this subject; but full light was thrown on it by Basedow[4] in 1840, who described a new kind of disease under the name of "Exophthalmic Cachexia" (Glotzaugencachexia), as the most prominent symptoms of which he names palpitation of the heart, goître, and exophthalmos. Since that time many cases have been published by different authors, of whom we need merely name Praël,[5] Gräfe,[6] Charcot,[7] Aran,[8] Trousseau,[9] Claude Bernard,[10] and Handfield Jones.[11]

SYMPTOMS.—The first symptom observed in these cases is the palpitation of the heart; at first slight, but rapidly increasing to a very great degree, and occurring in paroxysms produced by slight exertions or nervous excitement. In the case published by Graves,[12] "the beating of the heart could be heard during the paroxysm at some distance from the bed, a phenomenon I had never before witnessed, and which strongly excited my attention and curiosity."

The number of beats is seldom less than 100, and may become so violent as to shake the whole thorax. Nevertheless, the physical examination of the chest can only detect organic alterations or irregularities of the rhythm when the disease has existed for a long time. In more advanced stages, however, dilatation of the heart takes place, and gives rise to all those changes in the peripheral circulation which usually accompany enlargement of one or both ventricles. It is a remarkable fact that even in cases in which the heart has become enlarged, a high degree of insufficiency of the valves is rarely met with, and yet systolic bruits and noises are common, and may be heard not only at the usual seats of such sounds, but also in the auricles and large vessels in the neck.

The irregularity in the heart's action is sometimes very marked, as

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[1] Robert J. Graves, M.D., A System of Clinical Medicine, p. 674, Dublin, 1843.
[2] Dublin Journal of Medical Science, vol. xx.
[3] Heidelberger Medicinische Annalen, 1837.
[4] Casper's Wochenschrift, 1840.
[5] Archiv für Ophthalmologie, Bd. iii. p. 199.
[6] Ibid. p. 278.
[7] Gazette Méd. de Paris, 1856, No. 38.
[8] Ibid. 1862.
[9] Ibid. 1862, No. 29.
[10] Gazette Hebdom. 1862, No. 37; and Gaz. Méd. de Paris, 1862, No. 37.
[11] Medical Times and Gazette, 1860, vol. ii. p. 541.
[12] Loc. cit. p. 674.

EXOPHTHALMIC GOÎTRE.

BY HERMANN BEIGEL, M.D.

SYNONYMS.—Grave's Disease; Basedow's' Disease; Struma Exophthalmica; Goître Exophthalmique; Tachycardia Strumosa (Lebert); Cardio-thyroid Exophthalmos (Walshe).

Under one of the above titles a group of symptoms has been described, of which the most constant are Exophthalmos, Goître, and Palpitations. Some authors[1] are of opinion that Flajani[2] had some notion of the connection existing between these symptoms, but after a careful examination of the numerous cases of exophthalmos which this author published, I have not been able to find anything confirming this opinion. It is true he mentions several times the occurrence of palpitation in cases of exophthalmos, but he is by no means conscious that the concurrence of these phenomena—he does not mention goître at all—constitutes a special disease. In another place he relates three cases of goître occurring with palpitation in men, and in all these he made a cure by mere local treatment of the enlarged thyroid gland; in these cases, however, he does not mention the existence of exophthalmos, so that we may safely say that Flajani was not aware of the disease at present known by the name of Exophthalmic Goître.

It seems that Percy[3] was the first to pay attention to the enlargement of the eyeball in connection with the other two symptoms, but he also did not recognize the real relation between them.

Graves[4] was the first who distinctly described three cases of palpitations and goître, and, when speaking of the latter symptom, he says that the enlargement of the thyroid "seems to be essentially different from goître in not attaining a size at all equal to that observed in the latter disease. Indeed, the enlargement deserves rather the name of hypertrophy, and is at once distinguishable from bronchocele by its becoming stationary just at the period of its development, when the growth of the latter begins to be accelerated. In fact, although the tumour is very observable when the attention is directed to it, yet it never amounts to actual deformity." We shall see when we come to the symptoms of the disease, that Dr. Graves' observations were perfectly correct, and still hold good. Besides these three cases he describes one[1] in which all three symptoms, viz., exophthalmos, goître, and palpitations were very marked. The latter developed first and produced a pulse "never under 120, and often much higher;" after about a year exophthalmos was noticed, and "in a few months the action of the heart continued with increasing violence, a tumour of a horseshoe shape appeared in the front of the throat, and exactly in the situation of the thyroid gland."

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[1] Dr. Paul, Berlin Klinisch. Wochenschrift, 1867, p. 227.

[2] Giusseppi Flajani, Collezione d'osservazioni e reflessioni di Chirurgie, vol. iii. p. 270, Roma, 1802.

[3] Caleb Hilliard Percy, M.D., A collection from the unpublished medical writings of. London, 1825.

[4] Robert J. Graves, M.D., A System of Clinical Medicine. Dublin, 1843, p. 674.

At about the same time Marsh[2] of Dublin and Pauli[3] treated of this subject; but full light was thrown on it by Basedow[4] in 1840, who described a new kind of disease under the name of "Exophthalmic Cachexia" (Glotzaugencachexia), as the most prominent symptoms of which he names palpitation of the heart, goître, and exophthalmos. Since that time many cases have been published by different authors, of whom we need merely name Praël,[5] Gräfe,[6] Charcot,[7] Aran,[8] Trousseau,[9] Claude Bernard,[10] and Handfield Jones.[11]

SYMPTOMS.—The first symptom observed in these cases is the palpitation of the heart; at first slight, but rapidly increasing to a very great degree, and occurring in paroxysms produced by slight exertions or nervous excitement. In the case published by Graves,[12] "the beating of the heart could be heard during the paroxysm at some distance from the bed, a phenomenon I had never before witnessed, and which strongly excited my attention and curiosity."

The number of beats is seldom less than 100, and may become so violent as to shake the whole thorax. Nevertheless, the physical examination of the chest can only detect organic alterations or irregularities of the rhythm when the disease has existed for a long time. In more advanced stages, however, dilatation of the heart takes place, and gives rise to all those changes in the peripheral circulation which usually accompany enlargement of one or both ventricles. It is a remarkable fact that even in cases in which the heart has become enlarged, a high degree of insufficiency of the valves is rarely met with, and yet systolic bruits and noises are common, and may be heard not only at the usual seats of such sounds, but also in the auricles and large vessels in the neck.

The irregularity in the heart's action is sometimes very marked, as in a case brought by Gräfe before the Berlin Medical Society, in which the disease was very intense, and this symptom strongly marked. The intervals between the single contractions in another case were so unequal, and the force of some contractions so slight, that no beat of the radial pulse corresponded to them, so that the beats of the heart exceeded those of the pulse by six or eight in the minute. Friedreich[1] has observed that in the more advanced stages, changes take place in the peripheral circulation similar to those which occur in the heart itself: so that not only the arteries of the extremities exhibit a full vibrating pulse, but that even in the smaller arteries, as for instance the metatarsal artery or the palmar arch, a throb may be felt.

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[1] Robert J. Graves, M.D., A System of Clinical Medicine, p. 674, Dublin, 1843.
[2] Dublin Journal of Medical Science, vol. xx.
[3] Heidelberger Medicinische Annalen, 1837.
[4] Casper's Wochenschrift, 1840.
[5] Archiv für Ophthalmologie, Bd. iii. p. 199.
[6] Ibid. p. 278.
[7] Gazette Méd. de Paris, 1856, No. 38.
[8] Ibid. 1862.
[9] Ibid. 1862, No. 29.
[10] Gazette Hebdom. 1862, No. 37; and Gaz. Méd. de Paris, 1862, No. 37.
[11] Medical Times and Gazette, 1860, vol. ii. p. 541.
[12] Loc. cit. p. 674.

The total number of beats may be even as many as 112, 120, or even 160 (Trousseau) in the minute; in three cases which I have recently observed, the pulse was in two of them never less than 120, and in the other, at a very early stage of the disease, not less than 100.

The second symptom is the enlargement of the thyroid gland. In some cases this may precede the palpitation, but the exophthalmos has never been observed as the first symptom. The enlargement is rather a swelling or fullness of the thyroid region than that kind of tumour which we observe in cases of true goître; but this rule has exceptions, and occasionally very large tumours may be observed. Generally one lobe of the gland is more affected than the other. A most peculiar appearance is sometimes produced by the trembling of the thyroid mass, combined with, or caused by, very intense throbbing of the carotid arteries; this phenomenon may be perceived by the eye as well as by the hand. Compression of the arteries does not materially diminish the size of the swelling. The surface is usually marked by large and tortuous veins, especially if the disease be of long standing.

Shortly after the commencement of the goître, and sometimes almost simultaneously with it, the eyeballs appear to become increased in size, and in many cases in a comparatively short time they protrude completely from the orbit, as if, indeed, they had almost fallen from their sockets. When the disease has reached so high a degree the eyelids are not capable of being closed, and sloughing, suppuration, or other changes in the course may occur. Tatham[2] observed a case of a girl of eighteen, who was very pale and of a strumous aspect; the eyes were so prominent that when the eyelids were closed as far as possible a portion of the cornea was still left uncovered; the conjunctiva was seen in a state of strumous chemosis, "there was a deep and extensive ulcer involving almost all the part of the cornea which corresponds to the upper segment of the iris, and the inner part of the surface was sloughing:" the sclerotic vessels could be seen in some parts, and were injected: there was much circumorbital pain."

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[1] Virchow's Pathologie und Thérapie, 2nd edition, 1867; Bd. v. 2te. abth. p. 313.

[2] Medical Times and Gazette, 1864, vol. i. p. 89.

In some cases the left eye has seemed more affected than the right one; the eyelids do not close even in sleep, and portions of the eyeball are thus constantly exposed to the irritations of the atmospheric air and particles of dust. The expression assumed by the patient's face is a peculiarly unpleasant and painful one.

In the less severe cases the patients usually consider that they are suffering merely from an affection of the eyes; and the symptoms exhibited by them consist, according to Gräfe, in a dryness and irritability of the whole conjunctiva, which is particularly noticed when the patient attempts to read. This is due solely to deficient falling of the upper eyelid, which is incapable of following the downward movement of the eyeball. As a later consequence of this condition, dilatation of the conjunctival veins occurs, and sometimes the formation of crusts—a process which Gräfe considers to be of a neuroparalytic nature, in which the trophic rather than the sensitive fibres of the trigeminal nerve are affected.

This imperfect falling of the upper eyelid when the patient is lowering the plane of vision, Gräfe considers, in the progressive stages of the disease under consideration, as pathognomonic, and as an additional constant symptom of the affection. He attempts to explain this phenomenon by supposing a convulsive action of the "Musculus Palpebralis Superior," which was discovered by H. Müller.[1] It consists of involuntary muscular fibres, and receives its innervation from motor fibres in the cervical part of the sympathetic. It lies under the anterior part of the levator palpebræ superioris, and is connected behind with that muscle; in front it extends as far as the upper margin of the tarsal cartilage. Its function is to assist the levator in raising the eyelid. Müller has also described a similar muscle in the lower eyelid, which is under the influence of the sympathetic.

With regard to impairment of vision, authors are divided in opinion; but the majority seem to be of opinion that there may be none. When it does occur, it seems to be due to the changes produced by the ophthalmia, and not to any primary changes in the refracting media. No abnormal appearances can be detected by the ophthalmoscope except dilatation and engorgement of the vessels. In two of the three cases which I have myself examined, the arterial pulse could be seen very distinctly in the retinal vessels. In the third case, staphyloma posticum existed in a very marked degree, and the patient was myopic; this affection had, however, existed many years before any symptoms of exophthalmos had commenced.

The general symptoms occurring in this disease are almost entirely related as effects to the three principal phenomena above described. The most frequent are fainting feelings dependent upon the palpitation of the heart, and a burning sensation in the face and all over the body, dependent upon the excited state of the peripheral circulation.

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[1] Verhandlungen der Physik. Medicin-Gesellschaft zu Würzburg, Bd. ix. 1859, p. 244; and Bd. x. 1860.

In consequence of the abnormal conditions of arterial tension, which have been described above, these patients suffer from noises in the head and ears, paroxysms of suffocative dyspnœa, very distressing thirst, and violent diarrhœa; sometimes even hæmoptysis may occur. The brain is likewise affected in cases of progressive development of the disease; thus Brück[1] describes the case of a young girl of 18 who exhibited occasional paroxysms of vertigo and mania; and Geigel's[2] patient, at the climax of the disease, was attacked by "Monomania de grandeur."

These are, however, extreme instances, and in many cases the symptoms are so slight, and remain for a long time so quiescent, that the patient only learns from his friends that his eyes are peculiarly large, and that his throat is increasing in size, without being himself aware of any other change. I had a young girl under my care at the Farringdon Dispensary, who was 18 years of age, and had always enjoyed good health. During the previous six months she had suffered frequently from palpitation of the heart and want of breath when moving quickly. A few weeks later she "fancied" that her neck was increasing; and again after a few weeks, her friends told her that her eyes were increasing in size; noises in the head were now felt occasionally, and the patient suffered from fainting feelings. She came under my care on the 26th of November, 1867, and I made the following notes of her condition:—She is strongly built and well nourished, but of a deadly paleness; the eyelids can be shut, although the eyeballs are remarkably prominent, of large size and glaring appearance. The left lobe of the thyroid gland is somewhat larger than the right; but the front part of the neck is decidedly fuller than natural. Artificial light cannot be borne. On auscultation and percussion, besides frequent action of the heart, nothing abnormal can be discovered. Pulse 117. All other functions of the body are natural. The patient had been employed in a warehouse as book-keeper, but was obliged to give up her situation in consequence of the disease. She was discharged on April 13, 1868, perfectly cured, and has since resumed her former occupation.

The treatment consisted in the employment of a mixture of digitalis and iron. In less fortunate cases the patient soon emaciates, the functions become disturbed, and in women irregularity of the catamenia is usually observed; general dropsy may subsequently occur, terminating in death.

ETIOLOGY.—Although we are aware that there are certain districts in which Goître is endemic, yet it is not known that the disease now under consideration is prevalent there. It has been generally observed in countries and in places where true goître is comparatively rare, and in persons who are not subject to enlargement of the thyroid gland. The disease generally attacks individuals between the ages of 20 and 30. Stokes has, however, observed it in a woman of 60; Trousseau in a boy of 14, and in a woman of 66; Rosenberg in a girl of 7; and Deval in a girl of 2½ years, after scarlet fever.

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Women are, it appears, more prone to the affection than men. Of twenty-seven cases collected by Rosenberg and Henoch, as many as twenty-three occurred in women; of Taylor's twenty-five cases, twenty were women; of the twenty-nine cases noted by Praël, only one occurred in a man. Of the eight cases published by Schnitzler, there were but two men. According to Gräfe, the relation of the number of males to females is as one to six; but the affection is stated by the same author to be more dangerous in men than women, both as regards the eye and the life of the individual.

Most of the patients are in an anæmic state, a condition which existed in all the three cases observed by myself; and generally, in the female patients, hæmorrhage goes hand in hand with the disease, or at all events irregularities of menstruation may be observed.

The development of the disease is more or less rapid, and often varies, without any assignable cause except general debility and other general phenomena. There are, however, a large number of cases on record which have been developed suddenly after mental and nervous excitement, exertion, or shock. As the effect of emotion, it has been seen occurring during a single night (Taylor and Trousseau); and also after a violent cough and vomiting (Adams); after the cold bath (Percy and Schnitzler); and after a blow on the head (Begbie and Gräfe). One case has been related by Gräfe, in which rapid development took place after forced coition.[1] The patient was a young man 22 years of age, who succeeded in satisfying his desires after a violent resistance of upwards of half an hour. Covered with sweat and prostrate with fatigue, he recovered his strength by taking several glasses of madeira; but on the following morning he felt much exhausted, and noticed a change in his appearance; after another day, exophthalmos was very marked, and, rapidly increasing, soon assumed a most terrible aspect. The first case of those published by Thomas Laycock[2] is very interesting, in so far as it has been rapidly developed after the patient had experienced an intense fright. And Trousseau relates the case of a lady who, after receiving the unexpected news of her father's sudden death, was attacked in a few hours by violent palpitation of the heart, considerable swelling of the thyroid gland and protrusion of both eyes.

It is evident from the foregoing cases that nervous affections are particularly liable to give rise to exophthalmic goître; but whether they can do so in otherwise healthy persons is rather doubtful; and it is more probable that the effect takes place in such individuals as are either of a highly excitable temperament, or reduced in their general health by loss of blood, long-continued diarrhœa, fever, insufficient nourishment, bad digestion, excessive mental work, or hysteria, &c. Mental emotions may produce the disease by the very intensity of their character. Thus Friedreich saw it occurring in a highly-gifted lady who was mourning for the sudden death of her son.

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[1] Berlin. Klinisch. Wochenschrift, 1867, p. 320.

[2] Med. Times and Gazette, 1864, vol. ii. p. 323.

From the cases at present published we cannot form any opinion as to the effect produced by hereditary influence which gives rise to this affection; and with regard to the influence of climate we also know nothing. It must, however, be borne in mind that the whole number of cases at present fully reported is comparatively small.

PATHOLOGICAL ANATOMY.—As we have just mentioned, the fully recorded cases are not numerous, and still more rarely have the post-mortem appearances been described. I agree, however, with the opinion expressed by Virchow[1]—that it is not only from the anatomical examination of these cases that we have to expect materials for forming an opinion on the nature of this disease, since the phenomena which constitute the first beginning of the disease are of more importance than the conditions observed in the later stages.

It has been shown above, that it is only when the symptoms of the disease have already existed for a considerable time that alterations of the heart occur, which are discoverable by physical examination. The same holds good with respect to the post-mortem appearances of that organ. In the first stage no alteration has been found; but at a later period it has been found dilated, the mitral valve incompetent, the walls of the ventricle generally thin, though in rare cases they may be thicker than normal. Marsh has reported a case in which a general dilatation of all parts of the heart existed; and other authors have described enlargement of one or both ventricles.

Other post-mortem conditions—such as hyperæmia of the spleen (Begbie and Banks), atheroma of the arteries, &c.—must be considered either as secondary results or as accidental occurrences.

The thyroid gland has been found enlarged by all authors. Virchow describes it in one case in which he made the examination as moderately enlarged, simply hyperplastic, without development of cysts, nodules, or gelatinous formations. The lobules of the gland were more prominent, the interstitial tissue abundant, and the veins enlarged, the arteries remaining unaffected.

Markham describes the glands as large and firm; and Basedow found in one case the enormously enlarged gland filled with hydatids and the veins throughout varicose. In one of Praël's cases the tumour weighed as much as a pound, and reached into the thoracic cavity;

[1] Krankhaften Geschwülste, Bd. iii. p. 73.

the right lobe was cartilaginously degenerated, and closely embraced the trachea.

The cause of the exophthalmos has not been shown by the post-mortem examination; and several theories have been devised to account for this condition. Basedow and Heusinger considered it to be a simple hyperplasia of the connective and fatty tissue situated in the orbital cavity; others, however, as Praël, have been unable to discover any such alteration. It has indeed been noticed by some authors that the eyeballs may return into their sockets after death, or may at last be replaced by very gentle pressure. Some have considered with Taylor, that this symptom was dependent upon hydrophthalmos, but this opinion has likewise been abandoned. Recklingshausen has seen the muscles of the bulb in a state of fatty degeneration; and Archibald Keith noticed that the veins of the orbit were filled with blood, and that some extravasation had taken place into the orbit. Neither of these appearances, however, which seem in some measure to explain the protrusion of the eyeball, has been shown to be constant in the disease under consideration, and still less to exist at an early stage of the affection, when the exophthalmos is already marked.

Of special interest is the condition of the cervical part of the sympathetic nerve, described by different authors. Some have found marked alteration in its structure; others have described the part in question as perfectly normal. Keith[1] describes the sympathetic nerves "on both sides, but especially on the left, as large; the middle and lower cervical ganglia of the left side much enlarged and very firm and hard; the middle cervical ganglion thick in length, varying in width from one-eighth to a quarter of an inch; the lower ganglion seven-eighths of an inch long, and nearly uniformly a quarter of an inch wide. The connecting cords were correspondingly enlarged. Of the branches, those proceeding from the middle cervical to the inferior thyroid artery, and those from the lower cervical to the vertebral artery, were much more enlarged than the remainder, which varied but little from the normal size. Under the microscope, the ganglia seemed loaded with granular matter, observing to a great extent the appearance of nerve-tubes and nerve-cells, and resembling more than anything else the aspect of a lymphatic gland in the early stage of tubercular deposit. . . . . On the right side, the middle and lower cervical ganglia, and especially the latter, were enlarged, firm, and hard, presenting appearances similar to those of the left side, but not to the same extent. The cellular tissue surrounding the ganglia of both sides was thickened and hardened."

Trousseau and Peter likewise found the cervical ganglia of the sympathetic materially altered in respect to the external appearances as well as of the microscopical; but Recklingshausen, on the other hand, found the trunk of that nerve, as well as the ganglia, rather smaller than natural, as if in a state of atrophy, but histologically not altered at all.

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[1] Med. Times and Gazette, 1865, vol. ii. p. 521.

In the case of Dr. Geigel, both sympathetic trunks were apparently thickened and enveloped in a fatty sheath of connective tissue; but the microscope could detect no alteration in the tissue of the nerves and ganglia, except an intense brown pigment present in the latter.

It is, therefore, necessary to bear in mind that alterations of the sympathetic nerves and their ganglia have been repeatedly observed, but that some authors have failed to detect any such alterations; and it must not be forgotten that physiology teaches us that changes may take place in nerves in such a manner as not to be discoverable by the microscope, the existence of such changes being rendered manifest by altered function in those parts supplied by the respective nerves.

NATURAL HISTORY.—Some few authors (Niemeyer, Duchek) have doubted the existence of Exophthalmic Goître as a distinct disease, considering the combination of the three principal symptoms as an accidental concurrence—an opinion, however, which is neither borne out by physiological observation, nor by the cases recorded in medical literature, and which is of too little weight to be seriously discussed.

Others (Basedow, Beau) have looked upon the disease as simply a cachectic condition; whilst others again (Henoch) tried to give a mechanical explanation of the phenomena, based on the increased activity of the heart, whereby a larger quantity of blood is pressed into the thyroid gland, and thus produces exophthalmos by venous stagnation and congestion. This theory is by no means borne out by observation, because in many cases the affection of the heart is not the first symptom; and in others, all three phenomena occur at the same time. Large numbers of cases of diseases of the heart, observed in different hospitals every year, are not connected with either goître or exophthalmos; while, on the other hand, in districts where goître is endemic, the other two symptoms—viz., palpitation and exophthalmos—do not occur; and thus the mechanical theory must be considered to be untenable.

The only theory which is capable of accounting for all the phenomena observed in these cases, is that which considers the disease to be dependent upon nervous influence; and the more recent writers (Aran, Trousseau, Friedreich, and particularly Geigel) have endeavoured to show that it is the sympathetic which is at fault. Trousseau's explanation presupposes irritation of the nerve: Aran traces the symptoms to irritation of the heart and vessels through the cervical part of the sympathetic; whilst Friedreich's opinion is, on the contrary, that there exists a paralysis of those vaso-motor nerves which exist in the sympathetic trunks. Dr. Geigel divides the symptoms, and attributes, on the one hand, the alteration of the vessels to paralysis of the vaso-motor nerves within the sympathetic, and on the other, the changes which take place in the eyeball, to the effect of increased irritation of the oculo-papillary motor twigs derived from the same source. Eulenberg and Landois draw the conclusion that the sympathetic must be affected from the frequency of suppuration of the cornea, the severity of which does not correspond to the other symptoms. Gräfe has observed this occurrence in ten men and four women, and in one case the whole cornea was completely destroyed.

In order to gain an insight into the question before us, we must examine the facts arrived at by several renowned experimentalists when irritating or dividing the cervical part of the sympathetic nerve. According to the unanimous opinion of Claude Bernard, Brown-Séquard, Remak, Schiff, and others, the following phenomena may be observed in the eye after division of the sympathetic in the neck. The eyeball retracts into the orbit; the membrana-nictitans is drawn forwards; the upper eyelid falls, and the lower is somewhat raised. If, however, the peripheral stump be galvanized, the phenomena are reversed; the eyeball protrudes considerably; the upper eyelid is slowly raised, and the membrana-nictitans retracts. When the trunk of the sympathetic is galvanized without being divided, the arteries become narrower, even to such a high degree as to be perfectly occluded.

The results of these experiments, though by no means sufficient to explain all the symptoms exhibited in Exophthalmic Goître, clearly point to nervous action as the real source of the phenomena.

The chief difficulty which meets us in comparing the symptoms of exophthalmic goître with the results obtained by dividing the cord of the sympathetic is, as Dr. Geigel justly remarks, that the ocular phenomena of the disease are exactly the reverse of those following the operation, and that galvanism is required in addition to produce exophthalmos. This result seems, in this instance at least, to be caused, as has been shown by Mosler and Landois,[3] by contraction of the musculus orbitalis discovered by H. Müller. These experimentalists have noticed the muscle to contract at each opening and closing of the current, thus producing a momentary protrusion of the eyeball in the dogs and rabbits operated upon. It was at one time believed that the orbital muscles could, by an irregular action, give rise to an exophthalmos; but it is clear that neither the recti nor the obliqui can have any such action; and therefore the discovery of the musculus orbitalis, and its connection with the sympathetic, has been of the greatest importance in enabling us to explain the influence of the nerve upon the eyeball.

The contents of the orbit exercise a more or less constant pressure upon the walls of the cavity, due in part to the active and passive movements of the bulb, and partly to the distensile force in the blood-vessels. To resist this pressure, there exists in most mammalia a

[1] Wien. Med. Wochenschrift, 1867, p. 1474.

[2] Berlin, Klin. Wochenschrift, 1861, No. 31.

[3] Centralblatt für die Medicin, Wissenschaft, 1868, No. 33.

the body, dependent upon the excited state of the peripheral circulation.

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[2] Med. Times and Gazette, 1864, vol. ii. p. 323.

From the cases at present published we cannot form any opinion as to the effect produced by hereditary influence which gives rise to this affection; and with regard to the influence of climate we also know nothing. It must, however, be borne in mind that the whole number of cases at present fully reported is comparatively small.

PATHOLOGICAL ANATOMY.—As we have just mentioned, the fully recorded cases are not numerous, and still more rarely have the post-mortem appearances been described. I agree, however, with the opinion expressed by Virchow[1]—that it is not only from the anatomical examination of these cases that we have to expect materials for forming an opinion on the nature of this disease, since the phenomena which constitute the first beginning of the disease are of more importance than the conditions observed in the later stages.

It has been shown above, that it is only when the symptoms of the disease have already existed for a considerable time that alterations of the heart occur, which are discoverable by physical examination. The same holds good with respect to the post-mortem appearances of that organ. In the first stage no alteration has been found; but at a later period it has been found dilated, the mitral valve incompetent, the walls of the ventricle generally thin, though in rare cases they may be thicker than normal. Marsh has reported a case in which a general dilatation of all parts of the heart existed; and other authors have described enlargement of one or both ventricles.

Other post-mortem conditions—such as hyperæmia of the spleen (Begbie and Banks), atheroma of the arteries, &c.—must be considered either as secondary results or as accidental occurrences.

The thyroid gland has been found enlarged by all authors. Virchow describes it in one case in which he made the examination as moderately enlarged, simply hyperplastic, without development of cysts, nodules, or gelatinous formations. The lobules of the gland were more prominent, the interstitial tissue abundant, and the veins enlarged, the arteries remaining unaffected.

Markham describes the glands as large and firm; and Basedow found in one case the enormously enlarged gland filled with hydatids and the veins throughout varicose. In one of Praël's cases the tumour weighed as much as a pound, and reached into the thoracic cavity;

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[1] Krankhaften Geschwülste, Bd. iii. p. 73.

the right lobe was cartilaginously degenerated, and closely embraced the trachea.

The cause of the exophthalmos has not been shown by the post-mortem examination; and several theories have been devised to account for this condition. Basedow and Heusinger considered it to be a simple hyperplasia of the connective and fatty tissue situated in the orbital cavity; others, however, as Praël, have been unable to discover any such alteration. It has indeed been noticed by some authors that the eyeballs may return into their sockets after death, or may at last be replaced by very gentle pressure. Some have considered with Taylor, that this symptom was dependent upon hydrophthalmos, but this opinion has likewise been abandoned. Recklingshausen has seen the muscles of the bulb in a state of fatty degeneration; and Archibald Keith noticed that the veins of the orbit were filled with blood, and that some extravasation had taken place into the orbit. Neither of these appearances, however, which seem in some measure to explain the protrusion of the eyeball, has been shown to be constant in the disease under consideration, and still less to exist at an early stage of the affection, when the exophthalmos is already marked.

Of special interest is the condition of the cervical part of the sympathetic nerve, described by different authors. Some have found marked alteration in its structure; others have described the part in question as perfectly normal. Keith[1] describes the sympathetic nerves "on both sides, but especially on the left, as large; the middle and lower cervical ganglia of the left side much enlarged and very firm and hard; the middle cervical ganglion thick in length, varying in width from one-eighth to a quarter of an inch; the lower ganglion seven-eighths of an inch long, and nearly uniformly a quarter of an inch wide. The connecting cords were correspondingly enlarged. Of the branches, those proceeding from the middle cervical to the inferior thyroid artery, and those from the lower cervical to the vertebral artery, were much more enlarged than the remainder, which varied but little from the normal size. Under the microscope, the ganglia seemed loaded with granular matter, observing to a great extent the appearance of nerve-tubes and nerve-cells, and resembling more than anything else the aspect of a lymphatic gland in the early stage of tubercular deposit. . . . . On the right side, the middle and lower cervical ganglia, and especially the latter, were enlarged, firm, and hard, presenting appearances similar to those of the left side, but not to the same extent. The cellular tissue surrounding the ganglia of both sides was thickened and hardened."

Trousseau and Peter likewise found the cervical ganglia of the sympathetic materially altered in respect to the external appearances as well as of the microscopical; but Recklingshausen, on the other hand, found the trunk of that nerve, as well as the ganglia, rather smaller than natural, as if in a state of atrophy, but histologically not altered at all.

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[1] Med. Times and Gazette, 1865, vol. ii. p. 521.

In the case of Dr. Geigel, both sympathetic trunks were apparently thickened and enveloped in a fatty sheath of connective tissue; but the microscope could detect no alteration in the tissue of the nerves and ganglia, except an intense brown pigment present in the latter.

It is, therefore, necessary to bear in mind that alterations of the sympathetic nerves and their ganglia have been repeatedly observed, but that some authors have failed to detect any such alterations; and it must not be forgotten that physiology teaches us that changes may take place in nerves in such a manner as not to be discoverable by the microscope, the existence of such changes being rendered manifest by altered function in those parts supplied by the respective nerves.

NATURAL HISTORY.—Some few authors (Niemeyer, Duchek) have doubted the existence of Exophthalmic Goître as a distinct disease, considering the combination of the three principal symptoms as an accidental concurrence—an opinion, however, which is neither borne out by physiological observation, nor by the cases recorded in medical literature, and which is of too little weight to be seriously discussed.

Others (Basedow, Beau) have looked upon the disease as simply a cachectic condition; whilst others again (Henoch) tried to give a mechanical explanation of the phenomena, based on the increased activity of the heart, whereby a larger quantity of blood is pressed into the thyroid gland, and thus produces exophthalmos by venous stagnation and congestion. This theory is by no means borne out by observation, because in many cases the affection of the heart is not the first symptom; and in others, all three phenomena occur at the same time. Large numbers of cases of diseases of the heart, observed in different hospitals every year, are not connected with either goître or exophthalmos; while, on the other hand, in districts where goître is endemic, the other two symptoms—viz., palpitation and exophthalmos—do not occur; and thus the mechanical theory must be considered to be untenable.

The only theory which is capable of accounting for all the phenomena observed in these cases, is that which considers the disease to be dependent upon nervous influence; and the more recent writers (Aran, Trousseau, Friedreich, and particularly Geigel) have endeavoured to show that it is the sympathetic which is at fault. Trousseau's explanation presupposes irritation of the nerve: Aran traces the symptoms to irritation of the heart and vessels through the cervical part of the sympathetic; whilst Friedreich's opinion is, on the contrary, that there exists a paralysis of those vaso-motor nerves which exist in the sympathetic trunks. Dr. Geigel divides the symptoms, and attributes, on the one hand, the alteration of the vessels to paralysis of the vaso-motor nerves within the sympathetic, and on the other, the changes which take place in the eyeball, to the effect of increased irritation of the oculo-papillary motor twigs derived from the same source. Eulenberg and Landois draw the conclusion that the sympathetic must be affected from the frequency of suppuration of the cornea, the severity of which does not correspond to the other symptoms. Gräfe has observed this occurrence in ten men and four women, and in one case the whole cornea was completely destroyed.

In order to gain an insight into the question before us, we must examine the facts arrived at by several renowned experimentalists when irritating or dividing the cervical part of the sympathetic nerve. According to the unanimous opinion of Claude Bernard, Brown-Séquard, Remak, Schiff, and others, the following phenomena may be observed in the eye after division of the sympathetic in the neck. The eyeball retracts into the orbit; the membrana-nictitans is drawn forwards; the upper eyelid falls, and the lower is somewhat raised. If, however, the peripheral stump be galvanized, the phenomena are reversed; the eyeball protrudes considerably; the upper eyelid is slowly raised, and the membrana-nictitans retracts. When the trunk of the sympathetic is galvanized without being divided, the arteries become narrower, even to such a high degree as to be perfectly occluded.

The results of these experiments, though by no means sufficient to explain all the symptoms exhibited in Exophthalmic Goître, clearly point to nervous action as the real source of the phenomena.

The chief difficulty which meets us in comparing the symptoms of exophthalmic goître with the results obtained by dividing the cord of the sympathetic is, as Dr. Geigel justly remarks, that the ocular phenomena of the disease are exactly the reverse of those following the operation, and that galvanism is required in addition to produce exophthalmos. This result seems, in this instance at least, to be caused, as has been shown by Mosler and Landois,[3] by contraction of the musculus orbitalis discovered by H. Müller. These experimentalists have noticed the muscle to contract at each opening and closing of the current, thus producing a momentary protrusion of the eyeball in the dogs and rabbits operated upon. It was at one time believed that the orbital muscles could, by an irregular action, give rise to an exophthalmos; but it is clear that neither the recti nor the obliqui can have any such action; and therefore the discovery of the musculus orbitalis, and its connection with the sympathetic, has been of the greatest importance in enabling us to explain the influence of the nerve upon the eyeball.

The contents of the orbit exercise a more or less constant pressure upon the walls of the cavity, due in part to the active and passive movements of the bulb, and partly to the distensile force in the blood-vessels. To resist this pressure, there exists in most mammalia a

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[1] Wien. Med. Wochenschrift, 1867, p. 1474.
[2] Berlin, Klin. Wochenschrift, 1861, No. 31.
[3] Centralblatt für die Medicin, Wissenschaft, 1868, No. 33.

membrane, which separates the orbit from the temporal fossa; and this membrane (membrana orbitalis) contains numerous unstriated muscular fibres. In man the orbit is limited by bony walls, not only towards the temporal fossa, but on all sides; the membrana orbitalis is, therefore, reduced to a small quantity of fibrous tissue, which partially fills up the spheno-maxillary fissure: this has been shown by Müller to contain involuntary muscular fibres, and serves as an antagonist to the other muscles of the eyeball. The discoverer is of opinion that the orbital muscle in man would, under ordinary circumstances, be incapable of protruding the eyeball; but the experiments of Mosler and Landois, just alluded to, show that it is capable of doing so in dogs and rabbits under the artificial stimulus of galvanism; and it is, therefore, possible that pathological changes may give rise to a similar result.

The symptoms referable to the heart and thyroid gland offer less difficulties to an explanation found on the influence of the sympathetic, in so much as their connection with the cervical ganglia are numerous and obvious.

It is the middle and lower ganglia to which we have to look for the morbid changes which influence the three parts principally affected in this disease; but we must wait for further facts which experimental physiology may bring to light before we can explain the nature of these changes. The cases in which post-mortem examinations have been performed, and in which no alteration of these parts was discoverable, do not militate against this view, as we have already alluded to the well-known fact that changes in the nerves may take place which are only to be recognised by the impairment of function, and not by change of structure.

PROGNOSIS.—Exophthalmic Goître must always be looked upon as a grave disease, and as we are at present entirely ignorant of the real origin of the affection, our prognosis must necessarily be somewhat uncertain. As a rule, however, the cause is a chronic one; and a large number of cases run their course for a considerable period without giving rise to any more serious symptoms than an occasional attack of faintness and of inability for active exertion. Even in the more advanced stages, symptomatic treatment can afford benefit, so that a patient who has previously been unable to do any work can undertake some lighter duties, without suffering serious inconvenience.

The most distressing, and unhappily very frequent complication, is the affection of the cornea, which results from its exposure to the atmospheric air. The advanced stages of the disease seem always to lead to a fatal result; but the statistics are unsatisfactory, in consequence of the small number of cases observed.

TREATMENT.—We have remarked that this disease occurs particularly in such people as are of weak constitution, or have suffered from loss of blood, or from an exhausted nervous system. It is, therefore, necessary to improve the general health by regulating their diet, giving them easily digested food, change of air or climate, and a course of iron, bark, and digitalis, either separately or combined.

In one case, Trousseau has seen an improvement following hydropathic treatment. Handfield Jones and Russell recommend strychnia. Friedreich has seen benefit obtained in serious cases by the continuous administration of large doses of quinine, 12 to 15 gr. in the twenty-four hours; and Duméril, Demarqué, and others, confirm this observation, and consider the drug to act directly upon the sympathetic. It is remarkable that, though many authors speak favourably of the use of iron, yet Bruch and Levy have seen patients get worse under the use of chalybeate spas.

The three cases observed by myself were treated almost exclusively by iron and digitalis. The one related above entirely recovered; the others have been doing well so far that the exophthalmos and goître have disappeared, and the palpitations and fainting fits, which existed in one patient particularly to a very distressing degree, have been reduced both in frequency and severity, so that the patient, who was formerly compelled to keep her bed, after a few weeks was able to do her house-work with comparative ease and comfort. At the time of her consulting me she was 38 years of age, unmarried, and had had palpitations since she was 7 years old, when she had the measles. These palpitations had increased in intensity during the last six years, and had been combined with want of breath when she exerted herself. The eyeballs had become prominent, and vision somewhat impaired. During the last twelve months she had noticed an enlargement in the thyroid gland, the right lobe of which was as large as a hen's egg when the patient first came under my care (November 9th, 1867). After some months it required careful examination in order to discover the still existing enlargement of that lobe. The third patient was 22 years of age, married, the mother of two children, and very anæmic. She had suffered from palpitation of the heart for four years before counselling me in 1867, which had increased on exertion, and had been particularly troublesome when she ascended stairs. The protrusion of the eyeballs was noticed by the patient and her friends about four months previously; but the enlargement ot the neck was so universal and inconsiderable, that the patient was not at all aware of it. She had suffered for two months before coming into the hospital from fainting fits occurring daily. She was under treatment during four months, when the symptoms had almost entirely subsided.

Aran recommends the application of ice to the cardiac region in cases in which the palpitation is very severe. If the goître should not diminish under general treatment, compression or iodine may be applied locally.

With regard to the exophthalmos, the patient must be recommended to wash his eyes frequently with tepid milk, to prevent the ocular conjunctiva from becoming irritated. Gräfe recommends pressure, applied directly to the bulb, in case of the eyelids still being able to close perfectly ; but at a later stage he recommends the operation of tarsoraphy, or detachment of the upper eyelid by an incision along its upper attachment. Compresses dipped in milk, or astringent or caustic solutions, may relieve the affection of the cornea if it should become troublesome.

DIABETES MELLITUS.

By T. Lauder Brunton, M.D., F.R.S.

Definition.—Diabetes Mellitus is a disease characterised by the persistent presence and excessive quanity of sugar in the urine. It is usually attended by dryness of the skin and mouth, thirst, increased appetite, loss of sexual power and desire, muscular weakness, and copious excretion of urine of high specific gravity. It induces emaciation, and death usually ensues from some pulmonary complication, such as phthisis, pneumonia, bronchitis or œdema, from carbuncles, or from acetonæmia.

History.—No distinct mention of the disease is to be found in Hippocrates, although certain passages in his writings have been cited as indicating his knowledge of it. Celsus describes it in clear terms, mentioning both the great increase of urine and the wasting of the body, which occur as its most prominent symptoms. The name Diabetes was probably first applied to it about the middle of the first century, A.D.; Aretæus and Galen being the first authors in which we find it thus described. Several of the writers of the middle ages notice it, but no distinct progress in the knowledge of the complaint was made until 1674, when Willis observed the sweet taste and smell of the urine, which he ascribed to the presence of sugar. Morton, in 1675, detected the same properties in the urine of diabetics. Previous writers had overlooked this, noting only the copiousness of the urine and the wasting of the body. Dobson, Pole, Home, and Cowley proved the correctness of Willis's ideas by actually separating the sugar from the urine.

In 1787 Rollo noticed that vegetable food was injurious in this disease, and introduced an exclusively animal diet as the mode of treatment. Up to this time most physicians had followed Galen, and regarded diabetes as a disease of the kidneys. Rollo's observations, however, showed that the older view of Aretæus, that it was due to disease of the stomach, was more nearly correct. In 1815 M. Chevreul demonstrated that diabetic sugar resembled that obtained from starch. In 1823 Tiedemann and Gmelin found that sugar is a normal product of the digestion of starchy substances; and Ambrosiani, in 1835, showed the presence of sugar in the blood. The researches of Dumas, Bouchardat, Liebig, and others proved that sugar could be changed into lactic acid by means of a ferment, and could be destroyed by means of alkalies. These observations gave rise to two theories; the one that sugar is formed with abnormal rapidity in the intestine, absorbed into the blood, and excreted in the urine; the other, that diabetes is due to imperfect destruction of the sugar, either in the intestine or in the blood. Some held that it underwent conversion into lactic acid as it was passing through the intestinal walls, while others believed it to be destroyed in the blood by means of the alkali therein contained.

It was at this point that the question was taken up by Claude Bernard. He tried to ascertain where the sugar was destroyed by giving it to animals in their food, and testing for it in the blood at various points in the circulation from the intestines onwards through the body. He thus found that when saccharine food was given sugar could be found in considerable quantities in the blood of the portal vein, the vena cava, and the right side of the heart; but that blood taken from the left side of the heart contained only traces of it. From this he concluded that it was absorbed from the intestines and carried by the blood to the lungs, where it was destroyed. But on repeating his experiments with animals deprived of sugar or starch and fed on flesh alone, he still found sugar in the blood taken from the right side of the heart, and on tracing this downwards along the vena cava he discovered that it had entered with the blood of the hepatic vein, which contained it in large quantities. It was also abundantly present in the liver, but it was almost completely absent from the blood of the portal vein, which contrasted very markedly in this respect with similar blood from animals on a saccharine diet. It was evident from these facts that the liver had the power of forming sugar, a function of the organ which had previously been unknown. By passing a current of water through the portal vein Bernard found that he could remove all the sugar from the liver, but when the organ was allowed to lie for some little time, sugar again appeared in it, and could again be extracted by washing. As the liver had been taken out of the body before this experiment was performed, it was clear that the formation of the sugar was a chemical and not a so-called vital process; and, as all the blood had been removed from the vessels by the water which had been injected into them, there could be no doubt that the sugar had not been formed from the blood which passed through the liver, but from some substance contained in the hepatic tissue itself. This substance was not diffusible like sugar, or it would have been washed out of the liver along with it. For a short time its nature remained unknown, but Bernard's attention had been attracted by the fact that a decoction of liver had a milky appearance, and this opalescence could not be removed by filtration. On adding alcohol to the decoction, a white flocculent precipitate was thrown down. This substance closely resembled starch in its chemical composition, and as it was readily transformed into sugar by boiling with dilute acids or by the action of various ferments such as vegetable diastase, saliva, or pancreatic juice, Bernard called it glycogen, and by this name it is now generally known. Hensen, of Kiel, discovered it independently almost at the same time as Bernard. Even before he succeeded in isolating glycogen Bernard noticed that the decoction obtained from the liver of an animal fed on sugar and starch was quite milky, while that got from an animal which had been fed on flesh alone was only slightly opalescent. This difference in opalescence indicated that in the former case the liver contained much, and in the latter little, glycogen. He also observed that the amount of sugar in the hepatic vein was nearly equal in both cases, although in the one the animal had got no sugar in its food, and in the other a great deal. He was thus led to regard the liver as having a second function, that of arresting sugar on its way from the intestine into the general circulation. As this function is performed by the conversion of the sugar into glycogen, he terms it the glycogenetic function; while the conversion of glycogen into sugar again is known as the glycogenic function. But his ideas have not been accepted unquestioned. First it was said that the liver does not form sugar at all. The chief supporters of this doctrine were Figuier and Sanson. Figuier thought that the sugar found in the blood of animals fed exclusively on flesh was derived from sugar contained in the flesh itself, and simply absorbed during digestion. Its apparent absence in the portal vein he attributed to its reactions being masked by some substances such as certain albuminous products of digestion which were contained in the portal blood and prevented its detection; but Bernard showed that notwithstanding the presence of these substances sugar could be found in the portal blood of animals fed on a mixed diet, and Dumas, that sugar could not be detected in it by fermentation when the diet consisted of flesh only. Sanson took up a somewhat similar position to Figuier, but with a little difference, necessitated by Bernard's discovery of glycogen. Instead of the glycogenic he attacked the glycogenetic doctrine, and instead of maintaining that the liver took sugar from the portal blood, and after retaining it a little while, gave it off again, he said that glycogen was only dextrine which the liver had abstracted from the portal blood and transformed a little more actively into sugar than other tissues could do. The other tissues, however, had a similar function, and this dextrine, which was a vegetable product, formed a constituent of blood and flesh, and thus during digestion found its way into the portal blood of carnivora. This objection was, however, refuted by Messrs. Bouley, Poggiale and Longet, who were appointed by the Académie de Médecine to inquire into it. They found no glycogen in ordinary butcher-meat, but obtained it largely from the livers of dogs fed upon it. They did not find it, however, in the muscles of such dogs. In the flesh of horses it was constantly found, and probably it was this fact which led M. Sanson, who had made his experiments in the veterinary school at Toulouse, to raise his objections to Bernard's theory. The most formidable opponent of

Bernard is Pavy, who was formerly his pupil and a strong supporter of his theory. On repeating Bernard's experiments he was struck by his failure to get anything more than traces of sugar from the blood of the right side of the heart; and at last began to think that the sugar-forming function of the liver was not exerted during life, but was a post-mortem phenomenon and only occurred under exceptional circumstances while the animal was alive. Pavy's observations regarding the small quantity of sugar present in blood drawn from the right side of the heart, and the almost entire absence of it from the liver when removed immediately after death, have been confirmed by many observers, Marcet, MacDonnel, Jaeger, Meissner, Ritter, Schiff, Tscherinoff, Eulenburg, Flint, and Lusk. It is to be remembered that by passing a stream of water through its blood-vessels Bernard succeeded in washing the whole of the sugar out of a liver after death, when, according to Pavy, the transformation of glycogen into sugar is going on most quickly. Flint has explained the absence of sugar from a liver during life, or one removed from an animal the instant it has been killed, and while circulation is still going on, by supposing that the great mass of blood which constantly passes through that organ washes out the sugar as fast as it is formed; but after death, or when the circulation is interfered with, the transformation of glycogenic matter into sugar goes on. The sugar is not removed under these conditions, and can then be detected in the liver. This explanation reconciles to a great extent the conflicting observations.

ETIOLOGY.—*Sex.*—This disease occurs more frequently among men than women in the proportion of about three to one, as appears from the annexed table of the numbers given by various observers. This proportion would of course be very greatly altered if the cases of glycosuria which occur during pregnancy and lactation were included, but these cases are omitted because they differ so much in symptoms and prognosis from true diabetes that they can hardly be classed with it. It must also be remembered that the urine of men is much more readily obtained by the physician than that of women, and that the disease may sometimes be overlooked in the female when it would have been detected in the male; but these cases cannot be sufficiently numerous to influence greatly the proportion already given:—

Name of Observer.	No. of Cases.	Men.	Women.
Griesinger . .	225	172	53
Zeminer . . .	62	49	13
Seegen . . .	140	100	40
Betz	31	24	7
Schäfer . . .	49	36	13
Leudet . . .	41	24	17
Schmitz . . .	104	77	27
Durand-Fardel .	334	256	78
	986	738	248

Age.—It occurs at all ages from childhood up to advanced old age, but is most common during adult life. The time of its appearance seems to be modified by the sex and condition in life of the patient. In a table given by Griesinger, the maximum number of cases occurred among men between thirty and forty years of age, whilst among women the greatest numbers were equally distributed between the two decades from ten to twenty and from twenty to thirty. Amongst the cases observed by M. Durand-Fardel the number of females affected by the disease between the ages of ten and twenty was greater than that of the males in the same decade as well as that of the females in the two succeeding ones. While amongst hospital patients it occurs most commonly between thirty and forty, it is found more commonly at a later period amongst persons of higher rank and easy circumstances; the decade from fifty to sixty being that of its maximum frequency. The following table represents the frequency at different ages in a total of 618 cases collected by different observers:—

Age.	Seegen.	Schäfer.	Leudet.	Schmitz.	Durand-Fardel.	Total.
0—10	—	3	—	1	—	4
10—20	5	11	—	8	10	34
20—30	23	4	2	9	11	49
30—40	21	7	2	16	37	83
40—50	23	5	6	16	92	142
50—60	43	1	12	38	101	195
60—70	14	2	10	12	47	95
70—80	1	—	8	4	2	15
80—90	—	—	1	—	—	1

Heredity.—The influence of heredity is not often marked, for while in some cases parents and children have both had this disease, in the great majority the children do not have it. But although it is not frequently hereditarily transmitted, the tendency to it may appear very strongly in a single generation, and several brothers or sisters in the same family may suffer from it. So much is this the case that Bouchardat states that his knowledge of the fact led him to discover the disease in a brother of one of his patients who had no suspicion that he was suffering from it.

Temperament.—The disease is very commonly met with amongst stout muscular and obese persons, often of a gouty habit, and especially those who have a tendency to accumulate omental fat. It also occurs, but more rarely, in an entirely different class of thin spare and active persons, with a tendency to neuralgia and other nervous troubles.

Habits.—Sedentary habits of life and free indulgence in the pleasures of the table appear favourable to the development of diabetes. It occurs most commonly amongst private gentlemen, priests, lawyers, and doctors; and is less frequent among men engaged in active bodily exercise.

Race seems to have some influence upon it, for while it is exceptional among negroes it is very common among Jews. This may, however, be simply due to the habits of the races, for while negroes are chiefly employed in manual labour, Jews generally engage in sedentary pursuits such as banking and commerce.

Climate.—It is difficult to estimate the influence of climate, because the diagnosis of the disease depends so much upon the medical men who reside in any particular country or district. If their medical training has been thoroughly good, they will recognise the disease when it will be passed over unnoticed by others, the deaths really due to it being ascribed to phthisis or other diseases merely consequent upon it. Moreover, the complaint will be influenced by other causes than that of climate, such as the food of the inhabitants, or their manner of life and general circumstances. It is said to be very rare in Holland, Russia, Brazil, and the Antilles, and more common in England, France, certain districts of Germany, as Thuringia and Wurtemberg, and of India, and especially in Ceylon. It is said to be very common in Normandy and in the agricultural districts of England.

Exciting Causes.—Amongst the exciting causes are injuries to the head and general shock, and occasionally also injuries to other parts of the body, nervous affections, especially tumours. Softening and degeneration of the brain or medulla oblongata seems also to have a powerful influence. The occurrence of the disease not unfrequently appears to date from violent and depressing emotions, and it is said to be more than usually frequent in times of great commercial depression, when such emotions are likely to be more generally prevalent. Excessive mental exertion, even when unaccompanied by worry, has sometimes been adduced as a cause, and some cases seem to arise from severe bodily exertion and sexual excesses. The influence of food is by no means certain, for although a large proportion of starch and sugar in food has been regarded, and apparently with great probability, as a cause of diabetes, yet it is not possible to produce the disease in animals by a rigid diet of this sort, and the number of persons who live almost exclusively on such a diet is very large. Nevertheless, the occurrence of the disease, especially in certain localities, would seem to favour the opinion; and so also does the fact that it has been observed to come on after a sudden change from an animal to a vegetable diet, and to disappear when the use of animal food was resumed. Exposure to cold, especially during the period of menstruation, seems to have brought it on in certain cases, and it has not seldom been observed to come on during the course of recovery from febrile diseases, especially during convalescence from ague.

SYMPTOMS.—The chief symptoms which induce patients to seek for medical advice in cases of diabetes are either increased thirst, commencing emaciation or muscular weakness while the appetite continues good or even excessive. Sometimes, too, attention has first been drawn to the disease by the existence of a cutaneous eruption, such as eczema of the vulva, or boils and carbuncles. The disease generally begins insidiously, and continues for a considerable time before those suffering from it think of seeking advice. Sometimes indeed it may continue for years without the patient's being aware of its existence, its long continuance being only ascertained by the patient's recollection that his urine had left white spots on his dress or on the ground during a certain period, he not being aware of the cause. At other times its onset is somewhat sudden, and excessive thirst or emaciation is observed to come on shortly after severe mental anxiety or work, after an injury or concussion, or after convalescence from some febrile disease. It has already been mentioned that two types of persons seem especially liable to diabetes, viz., the stout, obese, and somewhat lymphatic, and the thin and nervous. It has seemed to the writer that the symptoms which indicate the commencement of the disease are different in these two types—thirst and emaciation being observed in those who belong to the lymphatic class, while muscular weakness and lack of energy first attract attention in those belonging to the nervous type. The number of cases he has as yet observed is too small to enable him to come to any definite conclusion concerning the uniformity or significance of this relation.

The symptoms of indigestion and nervous derangement have been mentioned as preceding the onset of the disease, or occurring during its very early stages, but it is difficult to say whether these are really part of the disease, and not rather its cause. The urine in diabetes is very greatly increased in quantity, often amounting to 80 or 100 ounces a day; it is generally of a pale colour, and a peculiar odour, differing from that of ordinary healthy urine, and something like that of whey. Its specific gravity is generally very high, usually ranging between 1030 and 1040, sometimes rising to 1045, or even 1050. It is often strongly acid. From the sugar it contains, it has sometimes a distinctly sweet taste, although occasionally this may be masked by its saline taste. A drop of it, falling upon the clothes or upon the ground, leaves, after evaporation, a white spot of sugar. When kept for several days, fungi are apt to grow upon it.

Although the increase in the quantity of urine is generally so great as to form a marked feature in the disease, yet sometimes its amount does not exceed the normal. The general symptoms are such as we should almost be led to expect from the drain of so much water and saccharine matter from the system. The thirst is generally very great, and the quantity of water drunk is often enormous. It has been said that the quantity of urine excreted by patients is greater than that of their beverages, but this can hardly be the case, and the observations which seem to support this view have probably been made for too short a time, the apparent excess being most likely derived from water lodged in the system. The thirst has been attributed to the necessity of water for the transformation of starch into sugar in the intestines, but it is rather due to the quantity carried out through the kidneys by the sugar in the process of excretion.

The author has observed that at least one diuretic, digitalis, has a similar power of causing thirst. Some years ago, when experimenting with this drug, and taking every day a measured quantity of fluid, the excretion of urine was greatly increased on one occasion by the use of digitalis. Shortly afterwards intense thirst came on, which obliged him to drink more than his usual allowance.

In addition to thirst, there is generally a dryness of the mouth. This has no direct relation to the thirst, and may even be present without it. This dryness in the mouth is more marked in the morning on awaking, and may be so severe as to interfere with articulation. The tongue is generally red, abnormally clean and fissured. Sometimes it is covered with a whitish and somewhat dry coat—sometimes a thin white fur shows itself in longitudinal streaks along the dorsum, and especially in the edges. Sometimes, however, the tongue is of a blackish colour, and may be dry and hard, or deeply fissured. Sometimes it is covered with large rough papillæ, and sometimes is sticky to the touch, seeming to the patient to adhere to the palate. The reaction of the saliva is generally acid; the gums are soft and somewhat livid, but sometimes pale. The teeth may become loose and seem to have been pushed upwards from the retraction of the gums. The odour of the breath is peculiar, and has been compared by some to hay, and by others to ripe apples. A disagreeable taste in the mouth is sometimes complained of, while by others the sweet taste of sugar is distinctly perceived. The appetite is generally increased, as one would expect from the necessity of supplying the body with nutriment to supply the want occasioned by the drain that is constantly kept up by the excretion of sugar. In some cases it is merely good, but not excessive, whilst in others it is perfectly ravenous. Towards the termination of the disease, when complications set it, digestion fails, and the appetite may sink under the normal, or disappear altogether. In some patients a great desire for fatty food has been noticed. The digestion is generally good, but as the disease continues, symptoms of dyspepsia such as flatulence, epigastric pain, and vomiting often occur. The bowels are frequently constipated. The skin is dry, wrinkled, and scaly in the advanced stages of the disease, but at its commencement the secretion of sweat may be normal or even excessive. The sweat is acid; it has the same odour as the breath, and has been found to contain sugar. Not unfrequently cutaneous eruptions of pustules or reddish or brown spots are observed on the body, thighs, and legs. Excoriation of the prepuce or eczema of the vulva frequently occurs from irritation of the parts by the saccharine urine. Although boils may occur at the very commencement of the disease, it is especially in the later stages that they and carbuncles become very common. The occurrence of either in a robust elderly man ought at once to lead to an examination of the urine. The lungs, at the beginning of the disease, are generally normal, except that the respiratory sounds are rather weak, especially towards the apices; but during the progress of the malady, exaggerated respiration, without any physical sign, has occasionally been observed, and very frequently the signs of pulmonary phthisis appear. The amount of carbonic acid excreted and of oxygen inhaled is less than usual, and it has been found by Pettenkofer and Voit that diabetic patients lose the power (which healthy persons possess) of storing up oxygen in the body during the night for utilization during the day. The general nutrition of the body may, as has already been observed, continue unimpaired for a number of years, but, in the advanced stages of the disease, even the increased appetite is insufficient to compensate for the constant loss of sugar, and emaciation occurs. One of the first symptoms of the disease is often loss of sexual appetite and power, and when this does not occur at the commencement, it generally comes on during the progress of the disease. Nervous disturbances are of frequent occurrence in diabetes, one of the most common being languor and incapacity for work, either bodily or mental, and there is frequently an irresistible desire to sleep after meals, which may, however, be due in a great measure to the sleep during the night being disturbed by the frequent calls to micturition occasioned by the profuse secretion of urine. The memory frequently fails, the patient becomes irascible, melancholic, and hypochondriacal. Pains are not unfrequently complained of in the lumbar region, in the back, legs, and joints, or chills or heat are felt in the extremities. Cramps in the legs are very common, especially at nights; hyperæsthesia is rare, but partial anæsthesia is sometimes noticed.

PROGRESS, DURATION, AND TERMINATION.—It has already been mentioned that in many cases the disease may exist for several years without the patient being aware of its existence, and its progress in such cases seems to be exceedingly slow—indeed, for a long time it may remain almost stationary, even although no special treatment be adopted. In the generality of cases it progresses steadily onward, unless it be arrested by appropriate treatment; the daily loss of nutritive material in the urine becomes greater and greater, until at length the increased appetite is insufficient to supply it, emaciation begins, complications set in, and death closes the scene. Intercurrent attacks of febrile disease diminish the amount of sugar excreted, but they do not exert much real influence upon the malady, and the sugar again increases when the temperature falls. The duration of the disease is usually long, although cases have been recorded in which it seemed to run a course of only a few weeks before phthisis set in. The usual term of the disease is said by Griesinger to be between two and three years. In many instances, however, when an appropriate treatment is rigidly pursued, its duration may be very greatly increased, so much so that, according to Bouchardat, a diabetic properly treated has as much chance of living a long time as a man in good health.

Death usually results from some complication of diabetes, the most common causes being phthisis, pneumonia, carbuncle, or gangrene.

Occasionally, however, diabetic persons rather suddenly become short of breath and drowsy, and the drowsiness rapidly deepens to coma, ending in death within a day or two of the onset of these symptoms. The peculiar condition suggests very strongly the action of a narcotic poison. It has been ascribed to uræmia, but all other evidence of uræmia is wanting, and a more plausible theory, in support of which much evidence has been accumulated, attributes it to the formation of a poisonous substance, acetone, by fermentation. The condition has therefore been termed "acetonæmia" (see Pathology).

COMPLICATIONS.—The complications of diabetes are such as arise from imperfect nutrition of the tissues showing itself in a tendency to inflame and slough. This tendency exhibits itself in the skin and subcutaneous cellular tissue, in the formation of boils, carbuncles, and superficial ulcerations upon various parts of the surface, and gangrene of the extremities resembling senile gangrene. The boils may appear in great numbers in the early stages of the disease before any of the other symptoms are present, and may first awaken suspicion regarding the nature of the disease. Carbuncles, on the other hand, generally come in the later stages, and are not unfrequently the immediate cause of death. Pneumonia, gangrene of the lung, and phthisis, often occur, the latter being the usual termination of diabetes. Disorders of vision are very common. Dimness of vision (amblyopia) occurs according to Bouchardat in about one case in five. It is generally transient, although it may recur. When it becomes permanent it is usually the forerunner of death. Cataract sometimes follows one or two attacks of temporary amblyopia, but sometimes appears without any previous failure of vision. It generally comes on after the disease has existed for some time, and may come on either gradually or quickly, and involve either one or both eyes. The author has seen it come on very shortly after the appearance of diabetic symptoms in a girl of fifteen years of age. It is nearly always of the soft kind.

DIAGNOSIS.—The diagnosis of diabetes depends upon the detection of grape sugar in the urine. The three conditions with which it may be most readily confounded are diabetes insipidus, temporary glycosuria, and hysteria. Diabetes Mellitus and Diabetes Insipidus resemble each other in the large quantity of urine passed by the patient, but in the former the urine contains grape sugar, while in the latter it does not. Glycosuria is much more likely to be confounded with diabetes, and, in fact, is constantly mistaken for it. The distinction between them is that glycosuria is a temporary, passing condition, and the quantity of sugar present in the urine is small. In diabetes, on the contrary, the sugar is permanently present, and is in considerable quantity. Glycosuria occurs almost as a normal condition in pregnant women, and sugar may occasionally be distinctly perceived in the urine after meals which consist of much saccharine and farinaceous food, although the persons are not diabetic. Sugar may also occur temporarily in the urine in cases of intermittent fever. In all those diseases the distinction between them and diabetes may be observed both by a continued examination of the urine, and by the absence of the other symptoms of the disease. The urine in them is not increased above the normal quantity; its specific gravity is not abnormally high, there is no emaciation, no excessive thirst, and no excessive appetite. Hysterical patients sometimes simulate diabetes, and the detection of the fraud is not always easy. They generally are unaware of the distinction between cane and grape sugar, and their urine, though sweet to the taste, and of abnormally high specific gravity, does not reduce Fehling's solution when boiled with it. If the urine, however, be first boiled for a few minutes with a drop or two of strong sulphuric acid, then neutralised with liquor potassæ and again boiled with Fehling's solution, reduction will take place, the cane-sugar having been converted into grape-sugar by boiling with the acid. A very instructive case has been recorded by Abeles. A lady came to Carlsbad complaining of diabetes, from which, as she alleged, she was suffering. On testing the urine in the manner just mentioned, it was found to contain cane-sugar, and not grape-sugar. On the physician informing the lady of his discovery of the imposition which she was practising, she left Carlsbad in great disgust, but returned the following year. On this occasion the specific gravity of the urine was as high as before, and it contained grape-sugar instead of cane-sugar. On testing the urine, however, by a polarising apparatus, Dr. Abeles found that the angle of polarisation corresponded with that of a solution of ordinary commercial grape-sugar, but not with that of diabetic sugar; and he intimated to the lady that he could only be satisfied regarding the nature of the disease by drawing a portion of the urine directly from the bladder for analysis. This she was willing to allow, but stipulated that it should be done at an hour to be fixed by herself. The urine which was drawn off by the catheter contained a very much larger proportion of grape-sugar than any which Dr. Abeles had previously analysed. The lady had actually gone to the trouble of introducing lumps of grape-sugar into the bladder. In consequence of the irritation thus produced, catarrh of the bladder came on.

PATHOLOGICAL ANATOMY.—The pathological appearances found on post-mortem examination of diabetic cases are by no means constant. They may be divided into two classes, those which may be supposed to have occasioned the disease, and those which may be regarded as consequent upon it. Amongst the former the appearance of the liver naturally first attracts attention. In many instances the organ appears quite normal. Not unfrequently, however, it appears enlarged and congested. The congestion is apparently equally diffused, has a light rosy tinge over the whole organ, and the acini appear, both in the natural surface and in the section, as sharply marked rosy specks, on which the fully-dilated capillaries can be recognised by means of a magnifying-glass as a thick network of red lines. The hepatic vein and its roots are not dilated. The enlargement of the liver is partly due to the congestion and partly to the enlargement of the glandular elements. The liver cells in the peripheral part of the acini are more rounded, the angles less sharp, the contents granular, and the nuclei large and distinct. Those in the middle part of the acinus which is supplied by the hepatic artery are fatty, those in the central district around the radicles of the hepatic vein are almost normal. In the periphery of the acini there seems to be an increase in their size, and also a formation of new cells, a microscopic section presenting large cells with several nuclei, and also young cells. The intercellular connective tissue is usually unaltered. At other times, however, it is increased, and the organ is dark and tough. Occasionally fatty degeneration has been observed. The pancreas is stated by Niemeyer to be not unfrequently hypertrophied, but according to other observers is oftener atrophied, the atrophy sometimes affecting the whole gland, and at other times only its tail, leaving the head of the gland unaltered. Cystic degeneration of the gland has also frequently been observed, and is possibly due to obstruction of a duct. In one case Seegen found fatty degeneration of the epithelium of the gland, and in another, obstruction of the duct by calculi, these being probably early conditions of the atrophy which occurs in the later stages. In the intestinal tract proliferation of the epithelium has been observed along almost its whole course, on the tongue, stomach, and intestine itself. The mucous membrane is thickened, and sometimes also the muscular coat. The surface is at times hyperæmic. The blood, as a rule, contains a larger quantity of fat than usual—so much so, that the serum sometimes appears milky. The heart is often fatty, and the vessels atheromatous, the small and moderately-sized vessels being more affected than the large ones. Those at the base of the brain and in the retina seem especially liable to atheroma.

The nervous system is very frequently the seat of lesions in diabetes, although these are neither very definite as to character nor seat. These may be conveniently arranged in physiological order, taking first the medulla oblongata, then the sympathetic and splanchnics, which contain the vaso-motor nerves to the liver, and then other parts of the nervous system.

The medulla oblongata has been found to be affected in a number of instances, sometimes by degeneration, sometimes by the pressure of a tumour, sometimes by clots or by inflammatory softening. Dickinson has noticed dilatation of the arteries and of the perivascular spaces. These are so large as to be perceptible to the naked eye, and contain blood more or less altered, according to the duration of the case. It appears to have left the vessels by diapedesis rather than extravasation. He considers this condition to be peculiar to diabetes, and has found it not only in the medulla oblongata, but in the pons and other parts of the brain, as well as the spinal cord. It has, however, been noticed in persons who were not diabetic, and has been found to be absent in some who were.

The sympathetic in the abdomen has been observed to be much thicker than normal, and the semilunar ganglion and splanchnics have been found thick and cartilaginous. Atrophy of the cells in the solar plexus has also been noticed. The vagi have been observed to be thickened in some instances, and in others atrophied from the pressure of concretion. The meninges of the brain have been found congested, adherent, or œdematous, and tuberculous deposits have also been discovered in them. Induration, hæmorrhagic infarcts, softening, and tumours have been observed in various parts of the brain, in the cerebellum, and in the pons. The spinal cord has in some cases been found hardened, in others softened; and Dickinson describes in it the same dilatation of the perivascular spaces as in the brain, and also enlargement of the central canal and proliferation of the epithelium lining it.

The kidneys are usually large and hyperæmic. The tubules show increased proliferation and often fatty degeneration of the epithelium. The lungs are generally diseased, though sometimes they present little or no change in cases where the disease has terminated in acetonæmia. The most usual alterations in the lungs are of a pneumonic character, portions of the lung being found in various stages of pneumonic change, red, grey, or ulcerated. The excavations are often large, and surrounded by thickened and indurated tissue. The lesions are thus chiefly those of chronic pneumonia, but true tuberculosis also occurs. Sometimes gangrene of the extremities is observed, and not unfrequently carbuncles have been found, which have made their appearance before death, and have indeed hastened the fatal termination. Inflammation of the aponeuroses, tendons, and bones also appears in some cases.

PATHOLOGICAL CHEMISTRY.—It may perhaps render the relation between the nature of the food and the formation of sugar in the body more comprehensible if we preface the consideration of this subject by a few words on the general relations of sugars and starches. These bodies are frequently termed carbo-hydrates, because they contain hydrogen and oxygen in the proportion to form water united with carbon. They may be divided into three classes—(1) sucroses, or the sugars proper; (2) glucoses, or grape sugars; (3) amyloses, or starch and woody fibre.

Each of these three classes contains several distinct substances having the same percentage composition:—

1. *Sucroses.*	2. *Glucoses.*	3. *Amyloses.*
$C_{12}H_{22}O_{11}$	$C_6H_{12}O_6$	$C_6H_{10}O_5$.
Sucrose +	Dextrose +	Starch +
(or cane sugar).	(or grape sugar).	Glycogen +
Lactose +	Levulose –	Dextrin +
(or milk sugar).	(or fruit sugar).	Inulin –
	Galactose –	Gums
	Inosite.	Cellulose.

Many of these bodies have the power of turning the plane of polarised light to the right or left, and this property is of considerable use in distinguishing them one from another. Those which turn the plane to the right hand are marked in the preceding table with +, those which turn to the left with a –.

The best known of the sugars is cane-sugar, or sucrose. It is readily soluble in water, and it has the power of rotating polarised light to the right, its power of rotation being 73·8. When its solutions are boiled for a long time alone, or for a short time after the addition of dilute sulphuric acid, it is split up by taking a molecule of water, and yields equal parts of dextrose and levulose, according to the following formula:

$$\underset{\text{Sucrose.}}{C_{12}H_{22}O_{11}} + H_2O = \underset{\text{Dextrose.}}{C_6H_{12}O_6} + \underset{\text{Levulose.}}{C_6H_{12}O_6}.$$

It does not ferment directly, but by the action of yeast it splits into dextrose and levulose in the same manner as when boiled with sulphuric acid, and these bodies are both capable of undergoing fermentation. When boiled with an alkaline solution of a salt of copper, it decomposes the copper salt slowly and imperfectly, while solution of dextrose does so rapidly.

Lactose, or milk-sugar, is less soluble than sucrose, and not so sweet. It rotates the polarised light in the same direction as sucrose, but to a less extent, its power of rotation being 59·3. When boiled with dilute acids, it yields a peculiar glucose called galactose.

The most important member of these groups in relation to diabetes is dextrose, or grape-sugar. It is readily soluble in water, from which it crystallizes in cauliflower-like masses; it is less sweet than cane-sugar. Its solution deflects the plane of polarised light to the right. When its solution is mixed with yeast, it rapidly undergoes fermentation, carbonic acid being given off, and alcohol remaining in the solution. It is decomposed, when in solution, by alkalies, the liquid becoming first yellow, and then brown. It is easily oxidisable, and therefore quickly reduces an alkaline solution of metallic salts, such as those of copper. Levulose resembles glucose in its reactions, but deflects the ray of polarised light to the left. This deflection to the left is much stronger than that of dextrose to the right, so that when equal quantities of these sugars are present in solution, deflection to the left occurs. This happens, for example, when cane-sugar is boiled with acids, or treated by the intestinal ferment, as it then splits up so as to yield equal quantities of dextrose and levulose. It is possible, however, to make a mixture of dextrose and levulose in such quantities that the rotatory powers will counteract each other, so that the solution will have no action on the polarised ray, although it contains a large quantity of sugar. Such a condition as this might occur in a diabetic patient after taking a quantity of cane-sugar, and this is probably the explanation of a case recorded some time ago, in which the urine had no effect upon the ray of polarised light, although it reduced copper strongly. When yeast is added to its solution, it undergoes fermentation, and splits up so as to yield chiefly alcohol and carbonic acid:

$$C_6H_{12}O_6 = 2C_2H_6O + 2CO_2.$$

Inosite is found in various animal tissues, and is thus often designated muscle sugar. It exists also in vegetables, and is obtained in considerable quantities from beans. It has no action on polarised light, and although it causes hydrated oxide of copper to resolve, it does not reduce it on boiling, nor does it undergo fermentation with yeast. In one case at least, observed by Kühne, and in which the urine was occasionally analysed by the writer, inosite appeared alternately with grape-sugar in the urine of a patient. In testing for inosite the urine is precipitated first with neutral acetate of lead, and then with basic acetate. The second precipitate is collected on a filter, suspended in a little water and decomposed by hydric sulphide. The solution is filtered and evaporated to a small bulk. A drop is then evaporated with nitric acid almost to dryness, on a piece of platinum foil. Ammonia and a solution of calcium chloride are added to the residue, and the whole gradually evaporated to dryness. If inosite be present a rose-red tinge will appear. It is still a question with some chemists whether another sugar should not be added to this group, viz., diabetic sugar; for although it is usually believed to be identical with glucose, this doctrine is not universally accepted, and it is quite possible that the progress of physiological chemistry may show that these sugars are distinct.

The physical properties of starch are well known. When heated with dilute sulphuric acid, or acted upon by certain ferments, as diastase, it divides into dextrin and dextrose:—

$$\underset{\text{Starch.}}{3(C_6H_{10}O_5)} + H_2O = \underset{\text{Dextrin.}}{2C_6H_{10}O_5} + \underset{\text{Dextrose.}}{C_6H_{12}O_6}.$$

Dextrin derives its name from its powerful action on polarised light, its rotatory power being + 138° 7′. It is generally prepared by heating starch to about 150 degrees. It is very soluble in water, from which it is precipitated by alcohol, and it is converted into dextrose by boiling with dilute acids. There are two varieties of dextrin, one of which gives no colour with iodine, and has therefore been named by Brücke achrodextrin; the other gives a wine-red colour with iodine, and is therefore called erythrodextrin.

Glycogen, when pure, forms a white, amorphous powder, dissolving in water, and giving an opalescent, milky solution. With iodine it gives a brown, red, or violet colour. It does not reduce an alkaline solution of hydrated oxide of copper, nor does it ferment with yeast. By boiling with diluted sulphuric acid, or by the action of diastatic ferments, such as diastase, ptyalin, or pancreatic ferment, it is converted into dextrose.

Tests for Sugar.—When sugar is burnt, it is converted into a substance called caramel, which has a peculiar distinctive odour. Upon this depends a simple test for its presence. A piece of blotting-paper soaked in the urine, dried, and held sufficiently near the fire or over a lamp to decompose the sugar, if it be present, will give the smell of caramel. This, however, is not very commonly employed. The four tests in general use are those of Moore, Trommer, Fehling, and the fermentation test.

Moore's Test.—In employing this test, equal parts of urine and of liquor potassæ are mixed in the test-tube, and the mixture heated to boiling. The sugar is thus decomposed, and yields glucic and melassic acids, which give a brown colour to the liquid, the tint varying in depth from a light brown almost to black, according to the amount of sugar. It is best not to boil the whole of the liquid, but only to heat the upper half of it, so that the colour of the lower half, remaining unchanged, serves as a standard wherewith to compare that of the upper. Any change in tint caused by the boiling is thus more readily detected.[1] The colouring matter of the urine also gives a yellow colour when boiled with potash. A high-coloured urine, therefore, may appear to contain a little sugar when it does not, and, to avoid this source of fallacy, the urine may be decolorised by shaking with animal charcoal and filtering. The yellow colour due to the action of potash on the colouring matter of the urine becomes lighter as the mixture cools. That due to sugar, on the contrary, becomes darker on cooling, and thus the one may be distinguished from the other.

Dr. Meymott Tidy has proposed to employ the method for quantitative analysis by means of a colour scale; but the method is not sufficiently accurate to be of use, except as affording a rough approximate indication of the amount of sugar present.

Trommer's Test.—This test depends upon the property which sugar possesses of de-oxidising metallic salts, so that when boiled with a solution of cupric hydrate it reduces it to cuprous oxide. In using this test, a drop or two of dilute solution of copper sulphate is added to the urine in sufficient quantity to give a faint but distinct blue colour to it, and then a quantity of liquor potassæ equal to that of the urine is mixed with it. If no sugar be present, a pale blue precipitate of cupric hydrate is thrown down, and does not dissolve on shaking; but if sugar be present, the precipitate re-dissolves, forming a rich blue solution. On then heating, the cupric hydrate is deoxidised, and a precipitate of yellow or orange-red cuprous oxide is thrown down.

Fehling's Test.—When the quantity of sugar is very small, it may happen that the whole of the cupric hydrate in Trommer's test may not be dissolved, and may interfere with the reaction. It is, therefore, convenient to have a liquid containing the cupric hydrate already dissolved. Several substances besides sugar cause cupric hydrate to dissolve in an alkaline solution, and amongst these is tartaric acid. Fehling's solution is of a deep blue colour, and when added to urine and boiled, gives a precipitate of cuprous oxide in the same way as in Trommer's test. The disadvantage of this solution is that it is apt to undergo change, and when old will deposit cuprous oxide, although no sugar be present. When using a solution, therefore, which has been made for some time, it is always well to boil it alone, to see that it does not deposit cuprous oxide, before using it as a test for sugar. Should it deposit when boiled, a little caustic soda or potash added to it will again render it fit for use.[1]

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[1] Dr. Gowers informs me that he has found that when a lemon-yellow tint is got with this test, the sugar contains about 5 grains to the ounce, 10 grains give a light sherry tint, 15 a dark sherry tint, and 20 or over a port-wine colour.

Fehling's solution is prepared by dissolving 34·64 grammes of pure crystallized cupric sulphate, powdered and dried between folds of blotting-paper, in 200 cc. of distilled water. In another vessel 173 grammes of Rochelle salts are dissolved in 480 cc. of a solution of caustic soda sp. gr. 1·14. The solutions are then mixed and diluted to one litre with distilled water.

Fermentation Test.—The easiest method of applying this is to mix the urine with a little brewer's yeast, and put it in a test-tube closed by a well-fitting cork, through which a piece of glass tubing passes nearly to the bottom of the test-tube. If the urine contain sugar, it will be decomposed by the yeast, and yield carbonic acid, which, if the tube be kept in an upright position, will accumulate under the cork, and by its pressure gradually drive the liquid out through the tube.

Several other qualitative tests for sugar have been proposed, but are comparatively rarely employed. Among others we may mention those of Böttger, Krause and Luton, Maumene, and Mulder.

Böttger's Test consists in mixing urine with a strong solution of carbonate of soda, adding a little sub-nitrate of bismuth and boiling the mixture. If sugar be present, the bismuth becomes grey or black.

Krause and Luton's Test.—Boil the urine with bichromate of potash and sulphuric acid, when, if the mixture becomes green or bluish-green, sugar is present.

Maumene's Test consists in steeping strips of woollen stuff in a solution of perchloride of iron, and drying them in a water-bath. If a drop of urine be put in one of these prepared strips, and heated over a spirit-lamp, a black spot forms if the urine contain sugar.

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[1] It is still better to keep the copper and alkaline tartrate separate, and only mix them when wanted, because, according to Dr. Gowers, Fehling's solution which has been made for some time will sometimes give a precipitate of oxide when boiled with healthy urine, although it will not when boiled with water. The solutions for this purpose may be made by dissolving the cupric sulphate alone in one litre of water and diluting the alkaline solution also to a litre. Another formula used by Mr. Martindale is 90½ grains of cupric sulphate dissolved in three fluid ounces of water for the cupric solution; for the alkaline solution 180 grains of caustic soda and 364 grains of tartrate of potash dissolved in three fluid ounces of water. When these solutions are mixed, they are equal to six ounces of ordinary Fehling's solution.

Mulder's Test.—A solution of indigo-carmine, rendered alkaline by carbonate of soda, is allowed to fall drop by drop into the urine, heated over a lamp. This solution gives a colour to the urine which is at first blue, but, if sugar be present, becomes green, reddish-purple, reddish-violet, and lastly yellow.

Quantitative Estimation of Sugar.—The specific gravity of the urine affords an indication of the amount of sugar it contains, but this indication is by no means exact, for the specific gravity merely indicates the quantity of solids in the urine, and not of sugar only; and as the urea in diabetes may be, and often is, greatly increased, it is evident that no reliance can be placed on the indications of the urinometer.

Therefore, as the specific gravity alone does not furnish a reliable indication of the quantity of sugar, it has been proposed to destroy the sugar by fermentation, and then calculate the proportion of sugar from the difference between the specific gravity before and after fermentation. Every degree of specific gravity has been shown by Roberts to correspond to about one grain of sugar per fluid ounce. Thus, if the specific gravity of the urine be 10·40 before fermentation and 10·20 afterwards, the sugar would amount to twenty grains per ounce.[1] The method consists in putting four ounces of the urine into a twelve-ounce bottle with a lump of German yeast about the size of a walnut, and stopping it with a cork, in which a nick has been cut to allow the carbonic acid to escape. A tightly-corked four-ounce bottle is filled with the same urine, but without any yeast, and both are then placed on the mantelpiece, or in some other warm place. The best temperature is 68° to 75° F., and it should be equably maintained. In about eighteen to twenty-four hours fermentation will be completed, the yeast deposits, the fluid clears, and is found on testing free from sugar. The specific gravity of the two urines may then be taken and compared. This process can easily be carried out by the patient. Another method is to ascertain the amount of carbonic acid generated from the sugar, but this is not a method of much practical use.

Fehling's Method.—The *rationale* of this method has already been described under the head of qualitative analysis of sugar. Its quantitative application depends on the fact that one atom of pure grape sugar = 180 is capable of reducing exactly five atoms = 397 of cupric oxide (CuO) to cuprous oxide (Cu_2O). If we know, then, how much oxide is reduced by a given quantity of urine, it is easy to estimate the amount of sugar it contains. This may be done by adding a solution of copper of known strength, drop by drop, to a measured quantity of urine, until reduction ceases, but in practice it is found more convenient to add the urine gradually to a known quantity of copper solution, boiling it after each addition until the whole of the blue colour is discharged. For the details of this process the reader is referred to special works on the analysis of urine.

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[1] According to Manassein the percentage amount of sugar is most readily got by multiplying the difference in specific gravity by 1,000 and dividing by 456. Thus in the case just given $\frac{20 \times 1000}{456} = 4{\cdot}3$ per cent.

Estimation by Polarisation.—The polariscope consists essentially of two Nicol's prisms, placed at a short distance from each other, one being fixed and the other being capable of rotation. When the axes of the prisms are parallel the light passes through freely, but when their axes are at right angles to each other they stop the passage of the light. If the plane of the ray polarised by the first prism is made to pass through a solution having the power of rotating it to the right or left while on its way to the second prism, the latter will no longer entirely cut off the light when placed at right angles with the first, and in order to stop the light it must be rotated to a greater or less extent according to the deflection which the ray has undergone during its passage through the fluid. When the second prism is attached to a scale, the angle of rotation is readily ascertained.[1]

PATHOLOGY.—The presence of sugar in the urine as an abnormal constituent has been long known; but the pathological processes which it indicates are only now, thanks to the labours of Bernard, Cyon, Eckhard, Flint, Lusk, Pavy, and others, beginning to be clearly understood. Even yet we generally find diabetes ranged in text-books with albuminuria and other diseases of the kidneys, although we now know that these organs are not at all in fault, but merely separate from the blood the excess of sugar present in it. The blood always contains more or less sugar;[2] but unless it amount to more than about one-third per cent.[3] it is not separated by the kidneys. Its appearance in the urine, therefore, indicates that an excessive quantity of it is present in the blood; and, if we can find out how this excess came to be there, we shall have discovered the pathology of glycosuria. An analysis of blood from the crural or jugular veins, and from the femoral and carotid arteries, shows that less sugar is contained in the venous than in the arterial blood; and therefore destruction of sugar must go on somewhere in the organism.[4] Excess of sugar in the blood may thus be due to two causes—(1) to increased introduction of sugar; or, (2) to diminished destruction of it; or to both of these together.

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[1] It is in this way that the instruments of Mitscherlich and Wild are made, but the most exact polariscope is that of Soleil and Ventzke, which is more complicated. In the latter, the rotation effected by a fluid is not ascertained by the lightness or darkness of the field of vision, but by the correspondence in colour of its two sides; and the rotation is ascertained, not by rotating a prism, but by turning a screw, which causes the two halves of a quartz compensator to slide upon one another.

[2] Bernard, Revue Scientifique, 2nd ser., tome iv. p. 1023. (Several of these lectures of Claude Bernard have been reproduced in the London Medical Record, 1873, and are well worthy of perusal, both for their matter and as admirable examples of method in experiment and clearness in diction.)

[3] Bernard, Physiologie Expérimentale, tome i. p. 302.

[4] Chauveau, Bull. de l'Acad., xxi., p. 1110, Sept. 1856. Harley, Brit. and For. Med. Chir. Rev. xxxix., July 1857. Bernard, Revue Scientifique, 2nd ser. tome iv. p. 1022.

We will first consider the ways in which an excess of sugar may be introduced into the blood; and this involves the question, How does sugar come to be present in it at all? The ultimate source of the sugar and of every other constituent of the body is, of course, the food we eat; and this, as we may easily see in a typical meal of beef-steak, bread, and pudding, consists of fat, albumen, starch, and cane-sugar. The fat takes no part in the production of sugar within the organism, but the other three do. After they have entered the intestinal canal, the starch is converted into grape-sugar by the saliva and pancreatic juice, and the cane-sugar into a mixture of glucose and levulose by the intestinal juice. The albumen is converted into peptones by the gastric and pancreatic juices. The sugar and peptones thus formed in the intestinal canal are absorbed by the intestinal veins,[1] but they are not all at once poured into the general circulation and carried to the brain and muscles. If this were the case, these structures would get all their nutriment at once, and they would have to stow it away themselves for use during the intervals of fasting. Possibly the reserve stores they would thus require to accumulate might clog their action; and, unless they could assimilate all the sugar at once, some of it would pass out in the urine, and thus be lost. It seems, at any rate, that such intermittent nutrition is not good for them, since a means of equalising it has been provided in the liver. This organ acts as a storehouse, in which the superfluous nutriment absorbed during digestion is laid up, and gradually given out again into the blood during fasting.

Glycogenetic Function of the Liver.—The sugar which has been absorbed from the intestines is conveyed by the portal vein to the liver; and there it is converted into glycogen, and stored up in the hepatic cells. How the transformation is effected we do not at present know; nor can we say whether the sugar is simply transformed into glycogen, or whether it splits up in some peculiar way so as to yield glycogen and fat. That glycogen is formed from sugar, and that sugar is stopped by the liver on its way from the intestine to the general circulation, are shown by several facts. In a starved animal, glycogen disappears entirely from the liver; but it quickly reappears if some sugar be injected into the stomach.[2] It is greatly diminished by fasting, and greatly increased by a full meal. When sugar is injected into the crural vein, it appears in large quantities in the urine: but when it is injected slowly into the portal vein, it is taken up by the liver, and not a trace of it is to be found in the urine. If the injection have been made too quickly, so that the liver cannot transform the sugar as rapidly as it is supplied, a portion of it passes into the general circulation and appears in the urine.[3] If the portal vein be ligatured, so that the blood finds its way from the intestines to the heart and body by means of the collateral circulation without passing through the liver, glycosuria occurs.[1] It would seem that it is only while sugar is being absorbed from the intestines that much of it is present in the portal blood, for generally there is less of it in the portal vein than of almost any other vessel in the body.[2] From the power of the liver to use up sugar in this way, Tscherinow has proposed to call its function glycophthiric, or sugar-destroying. Peptones also form glycogen, for the liver of a dog always yields a much greater quantity of this substance after the animal has had a meal of flesh than when it is fasting. Fick has advanced the hypothesis that the peptones which result from the digestion of albuminous food in the stomach are not again built up into albuminous substances in the organism, as is usually supposed. Instead of this, they undergo further decomposition, and split up into non-nitrogenous and nitrogenous bodies. The non-nitrogenous products are stored up in the body, and serve it as fuel; while the nitrogenous ones are excreted in the form of urea.[3] The urea is probably not formed directly from peptones; for, when they are injected into the blood, the urea is not increased till four or five hours after the injection; and, when meat is digested in the stomach, the increase of urea begins five or six hours after the meal.[4] It is probable, from Schultzen's researches,[5] that glycocine, leucine, and tyrosine, are first formed, and that these afterwards become converted into urea. Fick does not say where he supposes the decomposition of peptones to take place; but I think we are not far wrong in saying that it probably does so in the liver and muscles.

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[1] Von Mering, Archiv. Anat. u. Physiol. 1877, p. 379.
[2] Tscherinow, Wiener Acad. Sitzber. Math.-Naturwiss., vol. li. Abth. 2.
[3] Schöpffer, Archiv. für Exper. Pathologie und Pharmacologie, vol. i. p. 71.

We may thus broadly say that albuminous food forms peptones in the stomach and intestines; and these are split up in the liver and muscles, so as to yield glycogen, a non-nitrogenous product, which is stored up in these organs, and urea which is excreted. This at once enables us to understand how it is that, in diabetic patients fed on animal food, the urea and sugar in the urine increase *pari passu*;[6] the sugar in them being in great part excreted with the urea, instead of being stored up as in the healthy body.

The first great function of the liver, then, is to form glycogen from the sugar and peptones supplied to it from the intestines, and to store them up till wanted. This is termed by Bernard its glycogenetic function.[7]

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[1] Bernard, Revue Scientifique, 2nd serie, tome iv. p. 1066. Compare also the analysis (Revue Scientif., tome iv. pp. 1108 and 1023); also Schiff, Untersuchungen über Zuckerbildung, 1859, p. 3.

[2] Kühne, Lehrbuch der physiologischen Chemie, p. 65. It is difficult to understand the conflicting statements of different observers on this point, except by supposing that their experiments were made on animals in different stages of digestion or fasting.

[3] Fick, Pflüger's Archiv, vol. iv. p. 40; also Rabuteau, L'Union Médicale, 1873, No. 107.

[4] Fick, Verhandlungen der physikal.-medicin. Gesellschaft zu Würzburg, new series, vol. ii. p. 53.

[5] Schultzen and Nencki, Zeitschrift für Biologie, vol. viii. p. 124.

[6] Ringer, Medico-Chirurgical Transactions, vol. xliii. p. 323.

[7] Revue Scientifique, 2nd ser., tome iv. p. 1155.

As the materials on which the liver acts are supplied to it by the *portal vein*, we may connect the *formation* of glycogen with this vessel; while, as I shall afterwards show, the *hepatic artery* is more closely related to its *destruction;* although, from the free anastomosis between them, neither vessel possesses any function exclusively. The portal vein in man has the power of contracting very considerably;[1] but the source of its vaso-motor nervous supply is not so well ascertained as that of the hepatic artery. We shall, however, return to this subject in another part of this article.

Diabetes from Deficient Conversion of Sugar into Glycogen.— We can at once see that, if the glycogenetic function be imperfectly performed, too much sugar will pass into the general circulation, and be excreted by the kidneys. It will only do this, however, during the time that the sugar produced by the digestion of starchy or saccharine food is being absorbed from the intestines; and the glycosuria arising from this cause will be intermittent, coming on after meals, and disappearing during fasting,[2] or when sugar and starch are excluded from the diet, and flesh only is used as food. It is evident that if the meals be taken so frequently that the whole of the sugar obtained from one cannot be excreted before absorption of the next one begins, the glycosuria will be remittent instead of intermittent. The sugar will be most abundant when digestion is at its height, and scantier when it is nearly finished; but it will never be entirely absent. This kind of glycosuria will be completely arrested by abstinence from farinaceous or saccharine articles of diet. According to Pavy, it is not uncommon among elderly people; and, so long as the urine is kept nearly free from sugar by attention to diet, it does not appear to injure their health.[3] This kind of glycosuria can be produced artificially in animals by ligature of the portal vein. If this vessel be occluded all at once the animal dies immediately, but if the ligature be tightened around it very gradually, death does not occur. The blood from the intestine then finds its way into the vena cava by way of the collateral circulation instead of passing through the liver; and any substance which may have been absorbed into it from the intestine is therefore carried directly into the general circulation without being exposed to any chance of alteration through the action of the liver upon it. Under such circumstances sugar appears in the urine of dogs which are fed on starchy or saccharine matter during digestion, and is absent while the animals are fasting. The sugar in these cases is found in the urine even when the quantity of starch or sugar given to the animal is small, the glycogenetic function of the liver being completely abolished. The same thing has been noticed by Dr. Colrat, of Lyons, and M. Couturier,[4] in patients suffering from occlusion of the portal vein in consequence of pylephlebitis or cirrhosis. Even in health, the power of the liver to remove sugar from the portal blood and convert it into glycogen is limited; and, if it be absorbed too quickly from the intestine, it will pass through the liver and appear in the urine. This occurs when sugar is taken in large quantities by men or by animals, unless it be prevented by some circumstance or other from being too rapidly absorbed.[1] Thus Bernard found that, when he injected a quantity of syrup into the stomach of a dog, sugar appeared in the urine. On repeating the experiment with a rabbit, no glycosuria was induced. The reason of this difference is, that the dog's stomach is generally empty, except immediately after a meal; and the syrup quickly passed into the intestine was altered by the intestinal juice, and was absorbed so rapidly that the liver could not convert it all into glycogen. The rabbit's stomach, on the contrary, always contains a large quantity of vegetable matter, even when the animal has been starved. This mass absorbs the syrup like a sponge, and only allows it to descend slowly into the intestine, so that digestion and absorption go on gradually, and the liver is able to convert all the sugar into glycogen.[2] Bernard shows in a very ingenious manner that the sugar which appears in the urine of the dog is really the same sugar which has been absorbed from the intestine and passed unchanged through the liver, and is not sugar derived from glycogen. The latter consists entirely of dextrose; but that obtained from the urine of the dog contains both dextrose and lævulose, which are the ordinary products of the digestion of cane-sugar by the intestinal juice.

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[1] Verhandlungen der physikal.-medicin. Gesellschaft zu Würzburg, 1854, p. 1.

[2] See Traube, Ueber die Gesetze der Zuckerausscheidung im Diabetes Mellitus, Virchow's Archiv, vol. iv. p. 118.

[3] Pavy on Diabetes, second edition, p. 144.

[4] Colrat, Lyon Médical, April 11th, 1875; Dr. Couturier, Thèse de Paris, 1875, No. 209.

The starch and cane-sugar contained in food are not usually converted into grape-sugar by the saliva, pancreatic and intestinal juices, so quickly as to supply sugar to the liver more rapidly than it can convert it into glycogen. An increase in the amount of any of these secretions, such as might be expected to occur in hypertrophy of the pancreas, for example, by leading to more rapid conversion of the food into sugar, might cause diabetes, presenting similar characters to that produced by incomplete glycogenesis in the liver. Almost the only distinction between them would be that, digestion being completed more rapidly in the latter sort, the secretion of saccharine urine would begin sooner after meals and last for a shorter time than in the former. To ascertain this, an examination of the urine would require to be made very frequently; and I am not aware that any observations of this sort are on record. Several cases of glycosuria apparently depending on such alterations in the liver or digestion as have just been described, and arrested by abstinence from starch and sugar, have been recorded by Camplin,[3] Parkes, Traube,[4] Rayer, Bence Jones, and others.

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[1] Pavy, op. cit., p. 141; and Vogel, in Virchow's Handbuch der speciellen Pathologie und Therapie, vol. vi. Abth. 2, p. 49.

[2] Revue Scientifique, second series, tome iv. p. 1066.

[3] Camplin on Diabetes; and Medico-Chirurgical Trans., vol. xxxviii. p. 69; also other cases quoted by Parkes in his work on Urine, p. 317.

[4] Traube, Virchow's Archiv, vol. iv.

In some of these cases, the failure of the liver to perform its glycogenetic function seems to have been the sole cause of diabetes; in them, it could be warded off for many years by abstinence from farinaceous food. In others, the failure of this function seems to have been quickly followed by other changes; and then abstinence from starch failed to prevent the appearance of sugar in the urine, although it had done so at the commencement of the disease. It is possible that another imperfect kind of glycogenesis sometimes occurs when the liver forms glycogen rapidly enough, but of such a kind as to be too readily broken up again. The glycogen obtained from the livers of animals is by no means always of the same quality; for, according to Kühne, some specimens are converted by ferments into sugar with great rapidity, while others are only changed by them after the lapse of hours, although these very specimens may be at once changed into sugar by boiling them with acids.[1]

It is to be observed that the liver is not the only organ in the body which contains glycogen, although it is the chief one. The muscles also contain this substance;[2] and it is found in considerable quantities wherever cell-growth is actively going on, as, for example, in fœtal structures, or in the inflamed parts of the lungs in pneumonia. It is possible that it is first formed in the liver, and is merely carried to these other parts; but as it is readily changed into sugar in the blood, it seems much more likely that muscles and young cells possess also, to some extent, glycogenetic powers, and that the glycogen they contain is actually formed by them from sugar, or peptones supplied to them by the blood. The sugar they receive may either be derived from glycogen in the liver, or directly from intestinal digestion; for it is almost certain that, although a great part of the sugar and peptones is stopped by the liver, the whole of them is not.

Diabetes, from imperfect glycogenesis, then, is to be ascribed chiefly to the liver; but the possible participation of the muscles is to be borne in mind,

Glycosuria during digestion may arise (1) from interference with the portal circulation, so that the blood passes into the vena cava by anastomotic vessels; (2) from imperfect performance of its glycogenetic function by the liver, either in consequence of its tissues being unable for the work they have to do, although the circulation is normal; or of dilatation of the hepatic vessels and too rapid a flow of blood through them; or (3) from the proportion of sugar in the portal blood being rendered too great by the ingestion of a large quantity of sugar into the intestine, by too rapid digestion of amylaceous food, or too rapid absorption from the intestine. It is recognised by the sugar appearing in the urine only during the digestion of starch or sugar, and being absent during fasting, or during the employment of an exclusively meat diet.

Glycogenic Functions of the Liver.—The second great function of the liver is to give out, during fasting, the nutriment which it has stored up during digestion. Although Pavy calls the glycogenic function of the liver a post-mortem phenomenon, it appears so quickly after death that the most rapid manipulation is required to prevent the appearance of sugar. It is very difficult to believe that the liver should in this respect die so quickly when we know that it will in another retain its vitality so long, and will go on secreting bile for two hours after its removal from the body, provided a current of blood be kept up artificially in its vessels.[1] There can be no doubt of the correctness of Pavy's observations, which have done much to increase our knowledge of this subject; but they do not overturn Bernard's theory of the functions of the liver, which may still be accepted as substantially correct. It is to Bernard also that we are chiefly indebted for our knowledge of the influence of the nervous system over the production of sugar by the liver. He had succeeded in stimulating the salivary glands to secrete saliva by irritating the roots of the fifth pair of nerves, and thinking to stimulate the liver in like manner to produce sugar by irritating the vagi, he punctured their roots in the fourth ventricle. As he expected, he produced glycosuria, but on cutting the trunks of the vagi and galvanising the peripheral parts, he caused no alteration in the production of sugar; but on irritating their central ends he greatly increased the formation of sugar.[2] This showed that the increase was not produced directly through the action of the vagi on the liver, but was a reflex phenomenon. The way in which it produced glycosuria seemed to be by dilating the vessels, and thus increasing the circulation through the liver.[3] The channels by which the nervous stimulus is conveyed to the liver have been determined by Bernard, Schiff, Eckhard, Pavy, and more exactly by Cyon and Aladoff.

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[1] Kühne, Lehrbuch der physiologischen Chemie, p. 63.

[2] Bernard, Kühne, and M'Donnell; Kühne's Lehrbuch der physiologischen Chemie, p. 307; Nasse, Pflüger's Archiv, vol. ii. p. 97.

The destruction of sugar which Bernard supposed to occur in the lungs was shown by Chauveau and Harley[4] to take place in the systemic capillaries; and the researches of Ludwig and his pupils, as well as of Schultzen and others, have done much to indicate the tissues where it is chiefly consumed and the exact method of its decomposition.

We may say that the nutriment stored up in the liver is again distributed by the glycogen, which has been stored up in the organ, becoming gradually transformed into sugar again. It is then washed out of the liver by the blood and carried with it into the general circulation. The conversion of glycogen into sugar is effected by means of a diastatic ferment, of which a minute quantity only is present in the liver itself, but which is contained to a much larger amount in the blood. When the flow of blood through the liver is slow, the transformation of glycogen goes on gradually, but it is quickened whenever the current becomes more rapid. It seems probable that, although the blood of the portal vein may have something to do with the transformation of glycogen into sugar, this process is more closely connected with the circulation through the hepatic artery; for those lesions of the nervous system which increase the flow of blood through the liver and induce diabetes, have, according to Cyon,[1] little influence over the calibre of the vein, but cause the artery to dilate widely. As the blood from the artery flows into the portal vein, any increase in the circulation within it also quickens that in the vein to some extent. The circulation in the liver may be increased either by raising the pressure of blood in the arteries generally, so that the blood flows more quickly through the hepatic artery, although its size may remain the same as before, or by causing it to dilate, so that it receives a greater share of blood, while the pressure in the arteries generally remains the same.

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[1] Ludwig and Schmulewisch, Ludwig's Arbeiten, 1868, p. 113.

[2] Bernard, Leçons sur le Diabète, p. 370, and Physiologie Expérimentale, tome i. p. 316.

[3] Bernard, Physiologie Expérimentale, tome i. p. 334.

[4] Chauveau, Bull. de l'Acad. xxi. p. 1110; Harley, Brit. and For. Med.-Chir. Rev. xxxix. July, 1857.

The pressure of blood in the arteries generally may be raised by compressing any large artery, by violent muscular efforts, or by interference with respiration. The operation of these causes is followed by a greater production of sugar in the liver, its increase in the blood of the vena cava, and even its appearance in the urine. Schiff was able to produce diabetes by ligaturing large vessels, and Pavy[2] found in his experiments that whenever an animal struggled either on account of discomfort or because its respiration was interfered with, the quantity of sugar in the blood of the vena cava and carotids was at once increased. According to Michea and Reynoso,[3] it appears temporarily in the urine after an epileptic or convulsive hysterical fit. This power of muscular action to increase the proportion of sugar in the blood is exceedingly interesting, for sugar is destroyed in the muscles during their contraction, and is in all probability to be regarded as at least a part of the fuel from which they derive their energy.[4] The action of muscles, which causes the destruction of sugar within them, at the same time leads to an additional supply being furnished to them, and thus the balance of waste and supply is properly sustained.[5] The occurrence of sugar in the urine of persons suffering from cholera, or who have died from exposure to cold, is probably also to be attributed, at least in part, to the contraction of the blood-vessels near the surface of the body increasing the circulation in the liver. The intermittent glycosuria which has been observed to be present during or after a fit of ague, and absent during the interval, may also be ascribed to the rise in the general blood-pressure which occurs during the paroxysm.

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[1] Cyon and Aladoff. Reprint from the Mélanges Biologiques (which Professor Cyon was kind enough to send to me); and Bulletin de l'Académie Impériale de Petersbourg, vol. viii. p. 91.

[2] Pavy, On Diabetes, pp. 62, 68, and 145.

[3] Trousseau, Clinique Médicale, ed. 2me, tome ii. p. 665.

[4] Ludwig and Genersich, Arbeiten aus der physiologischen Anstalt zu Leipzig, 1871, p. 75.

[5] Ludwig and his pupils, *vide* Ludwig's Arbeiten, 1876, p. 45.

Dilatation of the hepatic vessels, and increased flow of blood through them, may be produced by paralysing their vaso-motor nerves either directly or reflexly. The vaso-motor centre (*a*, Fig. 1) is situated in the medulla oblongata. From it proceed those constant stimuli to all the vessels in the body by which their state of moderate contraction, or tone, as it is often termed, is maintained. The vaso-motor nerves for the hepatic vessels, indicated in the figures by the dotted line which accompanies them, pass from the vaso-motor centre down the spinal cord for a certain distance, then proceed through some of the communicating branches to the sympathetic cord and through the splanchnic nerves to the liver. The point at which they leave the spinal cord and pass to the sympathetic is not quite certain. According to Cyon and Aladoff[1] they leave the cord by means of the fibres (*c*) which accompany the vertebral artery, passing in them to the lower cervical ganglia. Thence they proceed in two fibres (*d*), one of which passes on either side of the subclavian artery, forming the annulus of Vieussens to the first dorsal ganglion (*e*), and thence through the gangliated cord of the sympathetic (*f*) and the splanchnics (*h*), to the cœliac ganglion (*i*), and along the hepatic vessels to the liver.

The first way in which dilatation of the hepatic vessels may be caused is by direct paralysis of the vaso-motor nerves for the liver. This may be produced by dividing these nerves at any point between the medulla and the liver. The influence of the vaso-motor centre will then be removed, and the hepatic vessels will dilate. Unless other circumstances should interfere, the flow of blood through the liver will be accelerated, and the production of sugar increased. Diabetes arising from division of the nerves is generally supposed to be permanent. In this it differs from diabetes induced reflexly by irritation of nerves, which ceases shortly after the irritation has passed off. This distinction is applied to that produced by operations; for, of course, permanent diabetes may depend on permanent irritation, and this is probably frequently the case where the disease occurs in man. Schiff[2] found that diabetes could be produced by division of the anterior columns of the spinal cord between the medulla and the fourth cervical vertebra. This lasted for days or weeks, in fact, till the animal died. Cyon and Aladoff have also observed diabetes which they attributed to paralysis of the vaso-motor nerves of the liver, after section of the fibres which accompany the vertebral artery, of the last cervical or first dorsal ganglion, or of the fibres forming the annulus of Vieussens. Eckhard has not succeeded in confirming their results,[3] and it is therefore by no means improbable that the vaso-motor nerves of the liver do not always leave the spinal cord to join the sympathetic by the fibres accompanying the vertebral artery, but sometimes pass further down the spinal cord, and leave it by the communicating branches going to some of the dorsal ganglia. This is all the more probable, as we have an instance of a similar kind in the case of the cardiac nerves.[1] As the vaso-motor nerves of the liver pass along the gangliated cord and the splanchnics, one would expect that section of these structures would

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[1] Cyon and Aladoff, op. cit.

[2] Schiff, Untersuchungen über Zuckerbildung in der Leber, 1859, p. 103.

[3] Eckhard, Beiträge, vol. vii. 1873, p. 19.

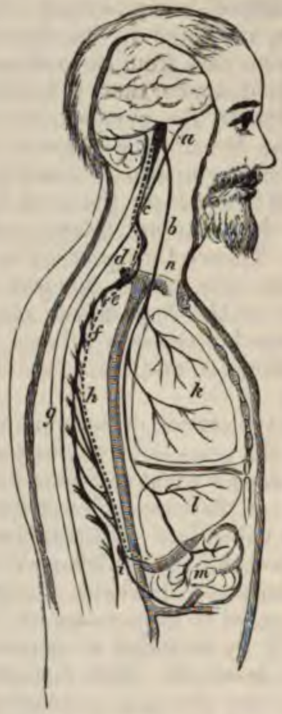

FIG. 1.—Diagram showing the course of the vaso-motor nerves of the liver, according to Cyon and Aladoff. These nerves are indicated by the dotted line which accompanies them : *a*, vaso-motor centre ; *b*, trunk of the vagus ; *c*, passage of the hepatic vaso-motor nerves from the cord along the vertebral artery ; *d*, fibres going on each side of the subclavian artery, and forming the annulus of Vieussens ; *e*, first dorsal ganglion ; *f*, gangliated cord of the sympathetic ; *g*, the spinal cord ; *h*, splanchnic nerves ; *i*, cœliac ganglion, from which vaso-motor fibres pass to the hepatic and intestinal vessels ; *k*, the lungs, to which fibres of the vagus are seen to be distributed ; *l*, the liver ; *m*, the intestine ; *n*, the arch of the aorta.

produce diabetes. But this is not the case, for the sympathetic cord may be divided between the tenth and twelfth ribs, or the splanchnics may be cut, without sugar appearing in the urine. On the contrary, when other means, such as puncture of the fourth ventricle, have been employed to induce it, no sugar appears in the urine if these nerves have been previously cut. At first sight, this result is very perplexing; but Cyon has most ingeniously explained it, by reminding us that it is not mere dilatation of the hepatic vessels, but increased circulation through them, which accelerates the formation of sugar; and the width of the vessels is of little consequence if there be not sufficient blood to fill them. Now, the vessels of the intestine, especially when the digestive canal is long as it is in rabbits, are so capacious, that when dilated they can hold as much blood as all the rest of the vascular system put together, and their vaso-motor nerves are also contained in the lower part of the cord and in the splanchnics. Consequently, when these are divided, the vaso-motor nerves of the intestinal vessels become paralysed as well as the hepatic ones, the vessels themselves dilate and retain so much blood that there is not enough left to increase the flow of blood through the liver, even though the hepatic vessels may be standing wide open to receive it. But if the vessels of the liver be first dilated, and the cord or splanchnics be then cut, the formation of sugar is not arrested; for a brisk circulation having once become established in the liver, still continues, although the intestinal vessels may become dilated.

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[1] Schmiedeberg, Ludwig's Arbeiten, vol. vi. p. 34.

A similar explanation may be given of the results obtained by Pavy, in one of his experiments.[1] He isolated the hepatic artery, portal vein, and hepatic duct, and then carefully divided all the remaining structures in the lesser omentum, including therefore the nerves passing to the liver. Notwithstanding that the hepatic nerves had been thus divided, and the splanchnics left uninjured, no sugar appeared in the urine. At first sight, this seems rather extraordinary, but a little reflection will show that the conditions are similar to those in Cyon's experiment. It is almost impossible to divide all the nerves of the liver without irritating the intestines and causing a considerable amount of congestion in them. Dilatation of the vessels produced in this way will have the same effect as division of the splanchnics. While watching a distinguished German physiologist dividing some of the nerves in the mesentery of a dog, I have seen the intestine become greatly congested, and so much blood has gone to it that there was not sufficient circulation in the brain to sustain its activity, and the dog, which was being operated on, slept soundly, although it had received no narcotic.

The second way in which the hepatic vessels may be dilated is by reflex paralysis, or inhibition, as it is generally termed, of their vaso-motor nerves. Every one knows that, when a sensory nerve is irritated, the impression is transmitted to the vaso-motor centre, and arrests its usual action over the vessels of the part to which the sensory nerve is distributed. Thus, when a grain of sand falls into the eye, the irritation which it occasions to the sensory nerves of the conjunctiva is conveyed by them to the vaso-motor centre, and arrests the action of that part of it which regulates the contraction of the conjunctival vessels. In consequence of this, they become dilated and full of blood, and continue so while the irritation continues; but, so soon as it is removed, the vaso-motor centre again regains its wonted power, and the vessels return to their normal size. The same is the case with the liver; and its sensory nerve is the pneumogastric. If this nerve be cut across, and its lower end irritated, no effect on the liver can be noticed; but, if its upper end be stimulated, the vessels of the liver dilate, the circulation increases, and sugar appears in the urine.[1] Irritation of the roots of the pneumogastric, in Bernard's famous experiment of puncture of the fourth ventricle, has the same effect as stimulation of its trunk. This experiment is performed by pushing an instrument like a brad-awl through the skull and cerebellum till it reaches the olivary fasciculi in the medulla oblongata. The instrument is prevented from injuring the anterior motor fibres of the medulla by a needle-like point which projects about the eighth of an inch from the middle of its edge. This is too fine to injure them itself, and, by coming in contact with the occipital bone, it prevents the edge of the awl from going too far. The irritation thus occasioned to the vagus roots inhibits the vaso-motor centre of the liver, and in half an hour or an hour, sugar appears in the urine. It does not remain more than a few hours, but disappears when the irritation has passed off; and in this it differs much from the diabetes produced by dividing the vaso-motor nerves of the liver. When this is done by cutting the anterior columns of the cord in the neck, sugar, as has been already mentioned, will persist for days or weeks.[2]

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[1] Pavy, op. cit., p. 171.

Irritation of the peripheral terminations of some branches of the pneumogastrics has a similar effect to one applied to their roots or trunks. These nerves are distributed to the liver, lungs, heart, stomach, intestines, &c., but these filaments do not all seem to have the same power of inducing dilatation of the vessels. Diabetes can be produced by irritating the liver by needles[3] or electrodes[4] placed in it, or by the injection of irritating drugs, such as alcohol, into the portal vein;[5] and irritation in the intestine seems occasionally to have the same effect, for there is a case on record of diabetes caused by the presence of a tapeworm in the intestines, and immediately cured by its removal. The influence of the cardiac, gastric, and other branches, has not been well ascertained. Eckhard has noticed the occurrence of diabetes after operations on the abdominal cavity without any definite lesion of nerves. The pulmonary branches seem to have a most important action, as the air inhaled during respiration appears to supply them with a constant stimulus, and thus continuously inhibits, to a certain extent, the vaso-motor nerves of the liver. When the pneumogastrics are cut across, this influence is of course removed, the hepatic vessels contract, and the production of sugar diminishes.

[1] Bernard, Physiologie Expérimentale, tome i. p. 326.
[2] Schiff, Untersuchungen über Zuckerbildung in der Leber, 1859, p. 108.
[3] Schiff, op. cit., p. 106.
[4] Pavy, op. cit., p. 137.
[5] G. Harley, British and Foreign Quarterly Review, July, 1857.

Increased respiration, on the other hand, seems to have the effect of increasing the formation of sugar; for Tieffenbach[1] observed that artifical respiration, performed by blowing air into the lungs by means of bellows, sometimes produced glycosuria. The appearance of sugar in the urine of animals poisoned by woorara is probably due partly to the artificial respiration employed to keep the animal alive, as well as to the action of the poison on the muscles, which will be considered afterwards. This is all the more probable because, when respiration is kept up to allow the animal to recover the effects of woorara, sugar makes its appearance. The fact that it does not do so while the animal is completely paralysed points to a reflex production of the phenomenon; for Schiff[2] has found that large doses of woorara paralyse the sensory as well as the motor nerves; and, as this loss of sensibility will destroy reflex inhibition, it is sufficient to explain the non-occurrence of reflex dilatation of the hepatic vessels while much woorara is present in the system. He states also that, when the respiration is effected gently, and the cannula is not tied into the trachea, but only laid loosely in it, so that the lungs are not over-distended by the force of the bellows, diabetes is not produced.[3] When the nerve-centres are very deeply narcotised, as when an animal is thoroughly under the influence of ether, even puncture of the fourth ventricle will not cause diabetes;[4] and Pavy[5] found no sugar in the urine of animals when he extirpated the superior cervical ganglion under the influence of chloroform, although it appeared when he narcotised the animals with the fumes of puff-ball, which, though it stupefied them, would probably not affect the vaso-motor centre through which the irritation of the operation would influence the hepatic nerves.

The glycosuria which is often observed after inhalation of carbonic oxide,[6] ether, and chloroform, may also be ascribed to irritation of the pulmonary branches of the vagus by these drugs before anæsthesia has become complete. If narcosis be quickly produced, sugar is not unfrequently absent; but it generally appears if narcosis be induced slowly, so that the irritant vapour acts long on the lungs, while the vaso-motor centre is yet unaffected by it. The glycosuria is not due to the struggles of the animal during the administration of the anæsthetic: for Schiff found it in a hedgehog which only succumbed to the influence of the anæsthetic after a long time, but did not make the slightest movement during the whole time.[7] The beneficial effect of opium and codeia in diabetes is probably due in part to their lessening reflex inhibition in the liver, as opium most assuredly does in the case of some other glands,[1] and thus diminishing the production of sugar. It seems probable also that reflex inhibition may follow irritation of other parts of the encephalon, and possibly also of sympathetic ganglia, as well as of the roots, trunks, and branches of cerebrospinal nerves.

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[1] Tieffenbach, Inaugural Dissertation; Königsberg, 1869. Abstracted in Centralblatt für die medicinischen Wissenchaften, 1869, p. 179.

[2] I am uncertain where this is published. Schiff himself informed me verbally of his discovery, and I have repeated his experiments with similar results.

[3] Schiff, Journal de l'Anatomie et de la Physiologie, 1866.

[4] Schiff, Untersuchungen über Zuckerbildung, p. 10.

[5] Pavy, On Diabetes.

[6] Schiff, Untersuchungen über Zuckerbildung, p. 124.

[7] Senff, Inaugural Dissertation, Dorpat, 1869; Henle und Meissner's Jahresbericht, 1869, p. 173.

The diabetes which has been observed after injuries of the cerebral lobes in man, of the cerebellum in animals by Eckhard,[2] of the superior cervical ganglion by Pavy, of the optic thalami, cerebral peduncles, pons Varolii, middle peduncles of the cerebellum, and of the cervical sympathetic cord and sciatic nerve by Schiff,[3] is probably due to this cause, as in all these cases it is only temporary, and not permanent. Irritation of the sciatic nerve in man seems to have the same effect as in animals, for temporary diabetes has been observed during an attack of sciatica.

There is another possible cause of increased formation of sugar in the liver; and that is, a greater proportion of diastatic ferment in it or in the blood which flows through it. The quantity of ferment in the liver of animals varies, and it is sometimes absent altogether. The same is probably the case with the blood. We do not know whether this ferment is peculiar to the blood, or whether it is ptyalin or pancreatic ferment absorbed from the intestines. Tiegel,[4] indeed, states that it differs from pancreatic ferment in being insoluble in glycerine; but von Wittich has obtained a diastatic ferment from the liver by glycerine, and I have done the same. It is possible that there may be two ferments having a similar action. Pepsin seems to be absorbed from the stomach, for it has been found in the muscles and even in the urine by Brücke; and pancreatic ferment seems also to be absorbed, for Hüfner found a ferment, possessing like it the properties of converting starch into sugar and of digesting fibrine, in the salivary glands and lungs. The increased secretion of a hypertrophied pancreas, which Niemeyer asserts to be common in diabetes, may thus not only act by quickening the conversion of starch into sugar in the intestines, but by increasing the transformation of glycogen after its absorption into the blood. But atrophy of the pancreas is quite as common a lesion in diabetes as hypertrophy; and if we suppose, as is usually done, that the only function of the pancreas is that of forming ferment, it is difficult to explain the occurrence of diabetes under these conditions, except on the supposition that in both of them the nerves of the organ are irritated, and cause reflex dilatation of the vessels of the liver in the same way that irritation of the liver itself does. But, if we suppose that the pancreas not only forms ferment, but also excretes ferment already circulating in the blood, in much the same way as the liver both forms and excretes bile, we at once see that the diastatic ferment which Hüfner supposes to be found in many parts of the body may accumulate in the blood because the pancreas can no longer excrete it, and thus lead to diabetes; for both bile and pancreatic juice can only be partially absorbed in the normal condition; another part will be evacuated with the fæces. I apprehend, however, that this explanation will seem to many persons to be too hypothetical, and that irritation of the pancreatic nerves and reflex dilatation of the hepatic vessels will appear a more probable cause of diabetes in cases of diseased pancreas. Plosz and Tiegel[1] have found that the ferment is contained in the blood-corpuscles alone, and not in the serum. When the blood-corpuscles are destroyed, the ferment acts vigorously,[2] as it is then set free. The diabetes observed by Harley after the injection of ether into the portal vein is probably due to the blood-corpuscles being dissolved by the ether.

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[1] In experiments upon the secretion of saliva I have found that in an animal deeply narcotised by means of opium, neither irritation of the lingual nerve nor chorda tympani causes the same marked dilatation of the vessels of the submaxillary gland which Bernard describes as occurring in animals which have not been narcotised.

[2] Eckhard, Pester medicin.-chirurgische Presse, No. 7, 1873, p. 98; and Hulz, Beiträge über Hydrurie und Mellituric, Marburg, 1872.

[3] Schiff, Journal de l'Anatomie et de la Physiologie, 1866, p. 376; and Untersuchungen über Zuckerbildung, p. 114.

[4] Tiegel, Pflüger's Archiv, vol. vi. p. 249.

I have already mentioned that the muscles contain glycogen, and, during their action, sugar is actually formed in them. The possibility of changes similar to those in the liver occurring in them must be borne in mind as a possible factor in diabetes.

Diminished Combustion of Sugar in the Body.—Having considered the effect of increased formation of sugar, we must now look at diminished combustion of it as a cause of diabetes.

The healthy organism is able to consume not only all the sugar produced within itself, but even more; and, if a solution of glucose or glycogen be injected in small quantities under the skin or even into the veins of an animal, no sugar will appear in the urine. This destruction of sugar probably goes on chiefly in the blood, lungs, and muscles, though it may take place in other tissues as well. Its occurrence in blood after it has been drawn is shown by the sugar contained in it disappearing after a short time when it is kept at a moderate temperature; and there is no reason to suppose that this does not go on within the body, more especially as Binz[4] and Zuntz have shown that the formation of acid, which goes hand in hand with the destruction of sugar, occurs even more quickly while the blood is still fluid, or, as we may say, alive, than after coagulation has taken place. The important part played by the lungs in the destructive process is evident from the great diminution which the sugar sometimes undergoes during its passage from the right side of the heart to the carotid artery; and the powers of muscle in this respect are shown by the observation of Ludwig and Genersich,[5] as well as of Bernard,[6] that the blood which passes through the vessels of a contracting muscle contains much less sugar when it issues from the vein than when it enters the artery. And yet, strangely enough, Ludwig and Scheremetjewski[1] found that, after grape-sugar had been injected into the veins of an animal, little or no increase took place in the oxygen consumed or the carbonic acid given off from the lungs—a result which indicates that grape-sugar, *as such*, is not burnt off in the body. On the other hand, however, they found that, when lactic acid as well as other organic acids, combined with soda, were injected into the veins, the amount both of oxygen and carbonic acid rose greatly, showing that these acids underwent combustion with great facility. Glycerine also undergoes combustion readily. Now, Bernard finds that, as the sugar disappears from blood, its place is taken by lactic acid;[2] and as Du Bois-Reymond has shown, an accumulation of sarcolactic acid in muscles occurs after they have been kept in action.[3] Muscles after death acquire an acid reaction by the formation within them of lactic acid from sugar or glycogen which they contain;[4] and when added to a solution of grape-sugar they will cause the formation of acid in it also. That this conversion of sugar into lactic acid is due to a ferment, is shown by the close correspondence between it and other processes of fermentation.[5] Thus, if the blood be heated so as to destroy the ferment, the sugar will remain unchanged;[6] and Binz and his scholars have shown that quinine and other substances, which lessen fermentation, likewise diminish the production of acid in blood. Bernard has also demonstrated that blood, like other ferments, acts more rapidly on glucose than levulose; whereas alkalies, to which the destruction of the sugar in the blood was long attributed, act more rapidly on the latter kind of sugar than on the former.

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[1] Pflüger's Archiv, 1873, vol. vii. p. 391.

[2] Tiegel, Pflüger's Archiv, vol. vi. p. 249.

[3] Tieffenbach, op. cit.

[4] Archiv für experimentelle Pathologie und Pharmacologie, vol. i. p. 20.

[5] Ludwig's Arbeiten, 1871, p. 75.

[6] Bernard, Revue Scientifique, 2nd series, tome iv. p. 1027.

The interesting experiments of Schultzen show more clearly than any others how sugar is decomposed in the body. A year or two ago, he found that, in animals poisoned by phosphorus, the processes of oxidation are arrested in the organism, but those of decomposition by ferments go on.[7] In such animals, urea disappears from the urine, and is replaced by leucine and tyrosine, which in the healthy organism are converted in urea.[8] No sugar appears in the urine, but a kind of lactic acid is found in quantities exactly proportional to the amount of sugar afforded to the animals by their food. This kind of lactic acid agrees exactly in its properties with the aldehyde of glycerine, and Schultzen considers the two bodies to be identical.[9] He thinks that, in the normal condition, sugar is split up by the action of a ferment into this glycerin-aldehyde and glycerine in the manner indicated in the following formula: Glucose $C^6H^{12}O^6$ + Hydrogen H^2 = Glycerin-aldehyde $C^3H^6O^3$ + Glycerine $C^3H^8O^3$. When the ferment is absent, as he supposes it to be in some cases of diabetes, the sugar is not split up, and so does not undergo combustion, but is excreted in the urine. In diabetics, the processes of oxidation are not impeded; for the nitrogenous waste products appear in the urine as urea, and not as leucine, as they do when oxidation is impaired in phosphorus-poisoning. The imperfect combustion of the sugar is due, he considers, to the want of ferment which should prepare it for oxidation, and not to the want of oxygen.

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[1] Ludwig's Arbeiten, 1869, pp. 144 and 141.

[2] Bernard, Revue Scientifique, 2nd series, tome iv. p. 1159.

[3] Du Bois-Reymond, Journal für Chemie, 1859, vol. lxxvii. p. 233.

[4] Bernard, op. cit., p. 1159.

[5] Some time ago I tried to separate this ferment from muscles by von Wittich's method, by glycerine, but was only partially successful.

[6] Bernard, Leçons au Collége de France. tome i. p. 230.

[7] Schultzen und Riess, Ueber acute Phosphorvergiftung, Separatabdruck aus den Annalen der Charité, Band x.

[8] Schultzen und Nencki, Zeitschrift für Biologie, vol. viii. p. 124.

[9] Schultzen, Berliner klinische Wochenschrift, 1872, No. 35. p. 417.

In view of these facts, we are, I think, justified in believing that the sugar which is present in the blood becomes converted by the aid of a ferment in the blood, muscles, and probably lungs also, into lactic acid and glycerine; and then undergoes combustion, thus sustaining the temperature of the body. Supposing, however, that this ferment is deficient, a greater or less proportion of the sugar will not undergo conversion into acid, and will then remain unconsumed, as in Ludwig and Scheremetjewski's experiment.[1] Unless the combustion of that quantity of sugar which does undergo transformation with the aid of fat, &c., be sufficient to sustain the temperature of the body, it will fall more or less below the normal; and this is actually what we find in a considerable number of diabetic patients. The muscular weakness which is observed in them is just what we should expect from the muscles being unable to make full use of the sugar which ought to supply them with energy for their work, though it is not improbable that excess of sugar in the blood may itself cause muscular fatigue. Whether this be the true explanation of their weakness or not, it is difficult to say; for we do not yet know how it is that chemical action is transformed in muscle into mechanical work; nor are we acquainted with the manner in which oxygen is stored up during the hours of rest in order to be expended during the time devoted to labour. Whenever we do learn this, we shall gain a deeper insight into diabetes; for Pettenkofer and Voit have found that patients afflicted with this disease have not the power, which a healthy man possesses, of absorbing more oxygen during the night than they need at the time, and using it up during the day. They are thus obliged to work as it were from hand to mouth, and are incapable of any great exertion.

The formation of lactic acid depends on the sugar as well as the ferment, and it is quite possible that the former may be in fault as well as the latter. I have already mentioned that different specimens of glycogen are acted upon by ferments with varying degrees of facility; and it would seem to be the same with sugar even when derived from similar sources, for Bernard, when arranging the different sorts according to the ease with which they are destroyed in the body, ranks sugar obtained from the liver above diabetic sugar.[1]

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[1] Ludwig and Scheremetjewski, op. cit., p. 145; Schultzen, op. cit.

The destruction of sugar probably goes on in the blood, brain, glands, &c., as well as in the muscles, but to a much less extent. In order that the sugar in the blood may be destroyed in the muscles, however, it must pass into them. Now, very little blood circulates through muscles when they are at rest, but the flow is much increased when they become active. More sugar will thus be carried to them and destroyed; but, as I have already mentioned, muscular exertion raises the blood-pressure, and increases the circulation in the liver and the formation of sugar in it, so that the balance is maintained. We can readily see that, if the liver go on producing sugar, and it be not destroyed in the muscles, it will accumulate in the blood, and at last appear in the urine. This, I believe, takes place in animals paralysed by woorara. Bernard thinks that the glycosuria produced by this poison, and also by large doses of morphia, is not due to their action on the muscles, but is to be ascribed entirely to their causing paralysis of the hepatic vaso-motor nerves and increased formation of sugar in the liver. I believe that woorara does exert this action; but the glycosuria is not due to it alone, but to its effects on the muscles also. My belief is founded on the experiments of Dock;[2] and, to make the grounds of it more intelligible, I shall shortly give the results of his research. 1. When rabbits are starved, glycogen disappears from the liver. 2. In such rabbits, puncture of the fourth ventricle does not produce diabetes. 3. After a few injections of cane-sugar into the stomach of starved rabbits, glycogen reappears in the liver. Injections of water, albumen, or fat, have not this effect. 4. If the fourth ventricle be punctured before the injection, no glycogen appears in the liver, and no sugar is found in the urine. 5. Poisoning by woorara produces diabetes in starved rabbits, although puncture of the fourth ventricle does not. 6. After poisoning by woorara, injections of sugar into the stomach do not produce glycogen in the liver; but sugar is abundantly found in the urine.

I must also mention that Weiss[3] has discovered that a considerable amount of glycogen remains in the muscles of starved animals after it has completely disappeared from the liver; and they retain their muscular activity as long as it is present in the muscles. The explanation of these results is by no means difficult. Indeed, it would have been easy for any believer in Bernard's theory of the causation of diabetes by puncture of the fourth ventricle to foretell that it would not induce glycosuria in animals when their livers contained no glycogen; for, this being absent, no increase in the hepatic circulation could increase the formation of sugar. When the puncture is made and the hepatic vessels are consequently dilated before cane-sugar is injected into the stomach, no glycogen is found in the liver; for it is converted into sugar, and washed away by the blood as soon as it is formed. It does not appear in the urine, for it is used up by the muscles as quickly as it is absorbed from the intestines. When the animals are poisoned by woorara, there is no accumulation of glycogen in the liver, for the poison paralyses the hepatic vessels, and thus produces the same effect as puncture; but it also prevents the muscles from using up the sugar, which therefore appears in the urine.

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[1] It is possible that this may be due to the sugar from diabetic urine consisting sometimes of a mixture of sugar derived from the liver and sugar absorbed from the intestine.

[2] Dock, Pflüger's Archiv, vol. v. p. 571.

[3] Weiss, Sitzungsberichte der Wiener Academie, vol. lxiv. p. 284.

The occurrence of sugar in the urine of starved animals after woorara-poisoning must be due to the glycogen in the muscles undergoing conversion into sugar, and its transformation being arrested at this stage, instead of changing into lactic acid and glycerine, and undergoing combustion as it ought to do.

There are some other substances, such as nitrite of amyl,[1] and nitrobenzol,[2] which probably cause diabetes chiefly by arresting the decomposition of sugar, although they may also act on the liver.

Since sugar has to be converted into lactic acid before it is burned off, and it is not improbable that the amount of this conversion is more or less regulated by the demand, we would not unnaturally expect that the injection of easily combustible organic acids into the blood, by preventing the combustion of sugar, might lead to its accumulation in the blood and its appearance in the urine. And such, in fact, is the case. According to Eckhard,[3] sugar appears in the urine of a rabbit after the introduction into its veins of carbonate, acetate, succinate, or valerianate of soda; and G. Goltz[4] noticed it after putting lactic acid into the stomach of the same animal.

To recapitulate shortly what has already been said: The liver has two functions—1, that of taking up the sugar which it receives from the intestines, and converting it into glycogen; and 2, that of forming sugar again from glycogen. The muscles probably possess three functions: 1, They take up sugar from the blood and convert it into glycogen; 2, They form sugar again from this glycogen; 3, They change both the sugar they form and the greatest part of that which they receive from the blood into lactic acid and glycerine, which undergo combustion.

Diabetes may arise—1*st. from increased formation of sugar*, due to (*a*) excessively rapid digestion of starch or sugar; (*b*) to failure or imperfection in the glycogenetic function of the liver, and possibly to some extent also of the muscles; (*c*) to increased transformation of glycogen into sugar, due to accelerated circulation through the liver, or a larger proportion of ferment in the organ or the blood. The circulation may be quickened either by increase of the general arterial pressure or by dilatation of the vessels of the liver, and especially of the hepatic artery. Increased blood-pressure may be due to muscular exertion, such as occurs in epilepsy, or to contraction of the arterioles, such as is caused by impeded respiration, exposure to cold, cholera, and Bright's Disease. The hepatic vessels may be dilated reflexly by irritation applied to the vagus, either at its ends in the lungs, liver, or intestine, in its trunk, or at its roots in the medulla, or to the cerebrum, cerebellum, pons, and probably some of the sympathetic ganglia. They may also be dilated, and the current in them accelerated, by section of their vaso-motor nerves at any point between the medulla and the liver, provided that the intestinal vaso-motor nerves are not also divided, and the supply of blood so much diminished that no increase in the hepatic circulation follows the section. Increased formation may also occur in the muscles.

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[1] Gamgee and Rutherford, quoted by Brunton in Sanderson's Handbook for the Physiological Laboratory, p. 515; and Hoffman, Reichert und Du Bois-Reymond's Archiv, 1872, p. 746.

[2] Ewald, Centralblatt der medicinischen Wissenschaften, 1873, p. 819.

[3] Eckhardt and Külz, Pesther medico-chirurgische Presse, Feb. 1873, p. 113.

[4] G. Goltz, Centralblatt für die medicinischen Wissenschaften, 1867, p. 705.

2nd. Diabetes may also arise *from lessened combustion*, due either (*a*) to insufficiency of the ferment which should convert the sugar into lactic acid and glycerine; (*b*) to an altered quality of the sugar which enables it to resist the action of the ferment; or (*c*) to diminished circulation through the muscles preventing the sugar from coming sufficiently into contact with the ferment.

PROGNOSIS.—The prognosis of diabetes depends considerably upon age; the younger the patient, the more unfavourable is it. Complete cure is rare, although almost every case can be very greatly benefited by treatment. When the disease occurs in patients under the age of twenty, few, if any, recover; but if it does not come on until middle age the prognosis is much better, and the patient may live for many years, even with little treatment.

The hereditary transmission of the disease adds to the gravity of the prognosis. It is much more serious when the onset is acute than when gradual, and when no apparent cause can be assigned than when it follows mechanical injury or mental anxiety. Its gravity also depends upon the amount of sugar passed, and on the relation of this to the digestive powers of the individual. If the quantity of sugar be great the danger to the patient is much increased, and if the digestive powers be insufficient to supply the drain upon the system, emaciation and death necessarily result. The drain is of course less when the quantity of urine is small, for even though it contain a considerable proportion of sugar, the total quantity passed daily is less than when the urine is copious. The absence of diuresis is therefore a favourable sign. When the patient is large, corpulent, and of a gouty diathesis, the disease may exist for a considerable time without producing much apparent effect upon the general health. The prognosis is much more favourable than in thin persons. Indeed, in some corpulent gouty individuals, the appearance of sugar in the urine about middle age would seem to deserve the name of gouty glycosuria rather than of diabetes, as it frequently gives rise to none of the usual symptoms of the disease except slight thirst. The ready disappearance of sugar on abstinence from starchy food is a favourable sign, but if sugar continues to appear when the patient is upon flesh diet, the case is much more serious.

The appearance of cataract is an unfavourable sign, and permanent amblyopia is frequently followed by a fatal termination. Carbuncles or gangrene are also of evil omen.

The appearance of albumen in the urine even when the quantity is slight is of very grave import, and when the appetite fails and diarrhœa sets in, or when the lungs show signs of consolidation or softening, there is little chance of recovery.

TREATMENT.—The most important item in the treatment of diabetic patients consists in careful attention to the diet. The excessive quantity of sugar, which in this disease is present in the blood, is not merely useless, it is positively injurious; and we must endeavour to reduce it as nearly as possible to the normal by causing the patient to abstain from those articles of food which produce it easily. These are—sugar of every kind, and in every shape; all foods containing starch, such as bread, pastry, puddings, maccaroni, vermicelli, rice, sago, tapioca, arrowroot, potatoes, beans, peas, carrots, parsnips, and turnips, as well as the softer and whiter parts of such vegetables as cabbage, Brussels sprouts, broccoli, cauliflower, asparagus, seakale. The patient must abstain from liver on account of the glycogen it contains, and must drink sparingly, even of milk, as milk sugar can be converted into a form of glucose. Sweet and sparking wines, and sweet ales, porter, stout, cider, liqueurs, should be avoided, and port wine only sparingly used. The diet may consist of any kind of flesh, whether of bird, beast, or fish, and whether fresh, dried, salted, smoked, or cured. Extracts of meat and gelatine may be taken in the form of soup, of meat extract, or of jellies. Eggs may be employed in any form, and so may fat, whether alone, as oil, butter, or mixed with albuminous matter, as in cream or cheese. Almonds and nuts of every kind, except chestnuts, may be freely indulged in. They contain much oil, and are very indigestible, but rather on account of their physical qualities than their chemical composition. Instead of being porous and spongy, like bread, and readily absorbing the digestive juices which thus act on every particle of the bread, they are tough and non-absorbent, and are generally broken by the teeth into small fragments, which are only attacked on their surface by the digestive juices. In order to get rid of this objection, Pavy suggested the employment of almond flour, and this is now made into a biscuit, by Mr. Blatchley. Wheat, in addition to starch, contains a quantity of gluten, and when the starch is washed away, this remains behind. Biscuits made of this have been suggested by Bouchardat, and are made in France, whence they are imported into this country.[1] Bran, also, when the starch has been washed from it, may be made into biscuits, and used instead of bread.

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[1] They are supplied by Bonthron, 106, Regent Street, and Van Abbot, 5, Princes Street, Cavendish Square.

Dr. W. Richardson, who himself suffered from diabetes, strongly recommends that the change from an ordinary to a restricted diet should be made very gradually, lest the patient become disgusted with his food. Rather than produce this injurious effect, it is better to relax the diet, and permit him to eat sparingly of bread made of whole meal, or even of white bread toasted and potatoes. Toasted bread seems to be less liable to cause sugar in the urine, although it is not easy to say why, unless it be that, being harder than untoasted bread, it is less rapidly acted on by the digestive fluids, so that the sugar it yields, instead of passing at once through the liver into the general circulation, is more gradually conveyed to this organ, and time thus afforded for its conversion into glycogen.

Such vegetables as consist almost entirely of cellulose, and contain little or no starch, as greens, spinach, cresses, and lettuce, may not only be freely used, but ought to form a considerable part of the food. Those portions of the plants which are green, consist chiefly of cellulose, and contain but little starch; the white parts of succulent plants, on the contrary, may contain a good deal, and had therefore best be avoided. The advantage of using green vegetables is that we not only enable the patient to vary his diet, and thus relieve the monotony of the diabetic regimen, but we give bulk to the food. It should not be forgotten that in Claude Bernard's experiments the sugar which he injected into the stomach of a dog appeared in the urine, but the same quantity did not cause glycosuria when introduced into the stomach of a rabbit, which is always more or less filled with undigested vegetable matter. By giving the patient, therefore, a quantity of insoluble vegetable matter along with those other parts of the food which will undergo digestion and afterwards pass to the liver, absorption of the soluble products of digestion will be more or less retarded, and more time will thus be given to the liver thoroughly to assimilate them. When patients, also, are put upon an almost exclusively flesh diet, the whole of it, or nearly so, being soluble in the intestinal juices, a very small residue remains behind, and the bowels are liable to become constipated. The addition of vegetables to the diet at once increases the bulk of the fæces, and tends to relieve the constipation from which patients might otherwise suffer.

With the view of supplying the place of the sugar lost in the urine, Piorry recommended the free administration of cane-sugar, but this treatment is wrong in theory and useless in practice. Dr. Donkin has advised a diet consisting exclusively of skimmed milk, six to eight pints of which are to be given daily for ten or twelve weeks. Few patients can stand this treatment for more than a week. Of those who can some die of inanition, others do recover. The recoveries are probably due to the semi-starvation producing benefits similar to those seen in the siege of Paris. Lactic acid has been given with good results in doses of about two drachms daily. When given in such large doses as three ounces daily it has produced symptoms of acute rheumatism. The author recommended some buttermilk in place of lactic acid some years ago, but he has found that it is impossible to obtain it in town, and that even in the country it is difficult now to get it.

The quantity of food consumed by diabetics should be carefully supervised, as there is a tendency to eat more than is wanted, and more than is likely to be good for them; for it must be remembered that although sugar, when present in excess in the blood, causes many disagreeable symptoms, including languor and muscular weariness, it is not the only substance which will have this effect. The products of the decomposition of flesh have a similar action in causing muscular fatigue. While, then, we put our patients upon a diet in which nitrogenous substances form the chief ingredient, in order, by this means, to prevent the formation of sugar in excess, we must be careful lest the patient take so much flesh that even the products of nitrogenous waste may undergo imperfect combustion, as well as the sugar, and pass out as urates or uric acid in the urine. Even when this is not the case, and when combustion is thoroughly carried on, the quantity of urea may be so great as seriously to tax the excreting powers of the kidneys. Moreover, every ounce of meat above what is required for the maintenance of the body proves injurious, not merely in the way that has just been mentioned, but by using up so much oxygen in the blood, and thus interfering with the oxidation of the non-nitrogenous substances of the body, sugar included. Bouchardat states that during the siege of Paris he observed sugar entirely disappear from the urine of diabetics in whom it had to that time persisted, even though they had been living on a carefully regulated diet. The diminution in the quantity of food, which the scarcity prevailing during the siege had occasioned, thus effected that which alteration in quality had failed to accomplish.

The quantity of water drunk must be regulated by the thirst. It is not advisable to stint the patient's supply so as to cause inconvenience. But the ingestion of large quantities of water by a healthy man may cause inosite to appear in the urine, and increases the excretion of urea which is already excessive in diabetes. The patient should therefore drink no more than is necessary to quench his thirst, and in order to do this more easily water acidulated with lemon-juice may be used instead of plain water. Water should not be drunk during meals, but should be taken in the intervals between them.

Next to diet, exercise is of chief importance. It should be taken, as much as possible, in the open air, and the great rule regarding it is, that the patient should take as much as he possibly can without *exhaustion*. So long as the exercise does not exhaust, it is beneficial, but beyond this point it is injurious. It may be taken in a variety of ways—walking, riding, athletic sports, or gymnastic exercises, according to the circumstances of the patient.

Dr. William Richardson, who gives his own experience of the beneficial effects of exercise in diabetes, says :—"Ten years ago, when I was first seized with an acute attack of diabetes, which threatened soon to end fatally, I became so weak, and had so little muscular power, that I could not walk a hundred yards without great fatigue. The muscles of my legs were so powerless that I fell two or three times; and in going down the slightest slope I had to pay unusual attention to my legs, or I was sure to fall. I began to take exercise regularly two or three times a day; wet or fine, I took it. Gradually I gained strength, so as to be able to walk five or six miles a day without fatigue. I now regularly walk from three to five or six miles a day." He also gives the case of a gentleman who derived but little benefit from a meat diet, Vichy water, iodide of potassium, or liquor arsenicalis, till he exchanged his sedentary life for active exercise, when his symptoms rapidly improved, and he soon recovered perfect health. The advice which Dr. Richardson gives regarding exercise seems to me to be so good that I take the liberty of quoting it. "The exercise should be regularly sustained day by day, even in wet weather it should not be intermitted; of course, great care should be taken against wet feet, and the shoes or boots ought to be changed in wet weather on returning home; it should never be carried to real fatigue; a feeling that exercise has been taken is the most that should be felt. To carry into effect regular and sustained daily exercise requires great moral courage and energy, the languor and feeling of weakness are so great; but if the exercise be only carried out patiently and perseveringly, the task will become not only more and more easy, but soon no longer a task, but positively a pleasure."

Bouchardat also insists very strongly on the necessity for exercise, despite the languor of the patient, and recommends gymnastic exercises, which are best performed in a gymnasium, both on account of the better direction which the master of the gymnasium may give to the exercises, and because the company of others prevents the tedium which is apt to be felt when they are carried on alone.

The clothing ought to be warm, as the patients have less heat-producing power than healthy persons, on account of their diminished oxidation, which has already been considered under Pathology. Great care should be taken to avoid cold, and, when the patient can afford it, residence in a warm climate may be advantageous. Warm baths are beneficial, and a Turkish bath should be occasionally used. Amongst the remedies, the most effective are opium and its alkaloids. Opium seems to have been given by Aretæus, in the form of theriaca Mithridatis, and it was employed in certain cases by Aëtius. It was recommended also by Paracelsus, Rollo, Willis, and others.

Under the influence of opium, the thirst diminishes, the excretion of urine becomes correspondingly less, and the proportion of sugar present in it falls. Morphia has a similar action, and codeia, first

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[1] Richardson, On Diabetes, p. 91.

recommended by Pavy, is even better, inasmuch as it can be given in large doses without producing drowsiness. Diabetics bear large, and sometimes enormous, doses of opium and codeia, and in administering both of these remedies it is well to push the dose until the sugar either disappears from the urine, or until increasing drowsiness obliges us to discontinue it. As much as thirty grains of opium, or several grains of morphia, and fifteen of codeia, have been given daily, not only without harm, but with great benefit. Senator recommends that the opium should be given in moderate doses from the very first, afterwards increasing until the excretion of urine is much diminished, when it should be discontinued for a time. He thinks it is best to reserve it for those occasions on which the diabetic, tired of a restrictive diet, returns to an admixture of farinaceous food. Both opium and its alkaloid codeia, however, may be frequently given continuously without harm. Other narcotics have not the same beneficial influence, although bromide of potassium has been found serviceable. Valerian is said by Lecorché to have an action similar to, though weaker than that of opium, and to be occasionally very useful in nervous cases. It should be administered in moderate doses, not exceeding thirty to fifty grains of extract in the day, given morning and evening for one or two months. Arsenic has been administered with varying results, sometimes having been of no service, at other times having been supposed to have effected cures. It has been more generally employed since the researches of Salkowski have shown that the livers of animals poisoned by it contain no glycogen, and that the puncture of the fourth ventricle in them does not cause glycosuria.

In cases of diabetes depending upon imperfect formation of glycogen in the liver, arsenic could hardly be expected to be useful, and it is possible that the different results obtained by different observers in some cases depend upon deficient glycogenetic, and in others on imperfect glycogenic function of the liver. It should be given in doses of from three to thirty drops of Fowler's solution three times a day, but care must be taken not to push it so as to interfere with the digestion. Tincture of iodine, in doses of twenty to thirty drops per diem, sometimes diminishes the sugar rapidly, but, like the liquor arsenicalis, it must be watched so as not to interfere with the digestion.

Alkaline remedies were introduced by Willis in the treatment of diabetes, and successive authors have since recommended them in various forms and for different reasons.

About thirty years ago, Mialhe employed them because he supposed that they would accelerate the decomposition and combustion of sugar in the organism. It is quite certain that they often prove very beneficial, but their mode of action is not clearly made out.

It is quite possible, as Mialhe supposed, they do increase the combustion of sugar in the blood, but it is also possible that they prove beneficial by lessening the formation of sugar in the liver. Pavy found that on injecting caustic alkalies or their carbonates into the portal vein, no sugar was found in the liver shortly after death. On removing the superior cervical ganglion sugar appeared in the urine of the animals experimented upon, but if he injected carbonate of soda into the jugular vein before the operation, no glycosuria was produced. He was first led to make this experiment by finding that caustic alkalies prevented saliva from converting glycogen into sugar. He thus supposed that it would also prevent the diastatic action of other ferments, and this idea has been confirmed by the experiments of Lomikowsky, who found that when bi-carbonate of soda was given to dogs for some time, little or no sugar was to be found in the livers, even when they were examined several hours after the death of the animals; but although sugar was absent, they contained a quantity of glycogen, and livers taken from other dogs to which no alkalies had been given, always contained sugar when examined in the same way. Alkalies may be given either made up as medicines in the usual manner, or in the form of natural mineral waters. Potash, soda, lithia, lime, and magnesia, or their carbonates may be given. All these have a local action upon the intestinal canal, in addition to the action which they exert upon the blood and tissues after their absorption. The salts which they form with organic acids, such as the citrates, tartrates, and acetates, have a less powerful action on the stomach and intestines, but after absorption into the blood the organic acid with which they are united undergoes combustion, and the salts just mentioned are all converted into carbonates and thus excreted in the urine, to which they give an alkaline reaction. In order to obtain a beneficial result, these medicines must be continued for several weeks or months, and it is therefore of importance to select a form which will interfere as little as possible with the digestion. For this purpose one of the best is the bi-carbonate of soda, which may be given in doses of from three to six drachms daily, in either pure or aërated water. The tartrate or citrate of soda may be given in somewhat larger doses, and Bouchardat recommends that it should be mixed with bread instead of common salt. The corresponding salts of potash may be given, like those of soda, but they are more apt to disagree with the stomach. The citrates and tartrates are not entirely converted into carbonates before their excretion in the urine by diabetic patients, when they remain at rest, as they are by healthy persons. When the diabetics are made to take a great deal of exercise in the open air, however, the greatest part of the citrates undergoes combustion in the body, and becomes converted into carbonates before excretion.

Carbonate of ammonia has been recommended by Pavy, in doses of from two to six drachms in a day. Lime-water may be substituted for potash or soda if the bowels should become loose, and magnesia if they should be constipated. Sulphate of soda has not been found to be of any service, but rather to increase the sugar in the urine.

The two chief watering places for the treatment of diabetics are Carlsbad and Vichy. At Carlsbad there are numerous springs; those which are chiefly used are the Sprudel and Mühlbrunnen. The quantity usually drunk is about six or eight glasses a day. Under this treatment the sugar in the urine diminishes or disappears, and the symptoms of the disease disappear in a corresponding degree. The waters of Vichy have a similar effect, and are used in very much the same way. From the beneficial effect of alkalies it might be supposed that acids would be injurious, and this, indeed, as a rule, they seem to be, although, if the digestion has been deranged by too long continuance of the alkalies, a short recourse to acids for a few days may not only be free from injury but productive of benefit.

Astringents sometimes diminish the amount of sugar, but do not seem to produce any marked benefit. The best of them, according to Bouchardat, is slightly sour claret. Diabetics are frequently more or less anæmic, and the administration of iron is advantageous. It may be given either alone in the form of the perchloride, carbonate, iodide, or hydrate, or in combination with bitter tonics. The author finds a combination of fifteen minims of perchloride of iron with hydrochlorate of morphia very useful. The morphia is given at first in doses of one-twelfth of a grain, and it may gradually be increased. When the more powerful preparations of iron disagree, the hydrate in the form of the dialysed iron will be found serviceable. Several ferments have been employed in the treatment of diabetes—yeast, pepsine, and raw meat. Yeast has been used by some with advantage, but in the hands of others its use has been followed by accidents, which might, or might not, have been due to the employment of the yeast, but which have rendered some chary of using it. Pepsine may be useful where there is derangement of the digestion, but it has little effect upon the progress of the disease. Raw meat was proposed some years ago by the author, with a view to supplying the ferment which converts sugar into lactic acid in the muscles, but the benefit actually derived from its use did not correspond with the expectations he had formed.

Treatment of the complications of the Disease.—The complications which arise in the course of diabetes require treatment somewhat modified by the presence of the disease itself. Cataract, so common in diabetic patients, is best treated by early operation. Neuralgias are not infrequent, and one might be tempted to employ subcutaneous injection of morphia for their relief, but in doing this it should always be remembered how prone any wound in a diabetic patient is to take on an unhealthy action. It is on this account that the use of the knife has been forbidden by some authors in the treatment of boils and carbuncles in diabetic patients, and it certainly seems advisable to do without it if possible. In the inflammations of internal organs to which diabetics are liable, stimulants must be freely employed, both because the patient is unable to utilise the nutriment which would in smaller inflammations be given to sustain the strength of non-diabetic patients, and because the latter are liable to asthenic forms of inflammation.

On account of the danger of acetonæmia occurring in diabetics who suffer at the same time from gastric catarrh the digestion ought to be carefully watched. To check the fermentative processes to which it owes its development, carbolic acid, salicylic acid, and thymol have been suggested, and during an attack it would seem advisable to empty the stomach by means of an emetic or stomach-pump, so as to prevent the absorption of any acetone which might be present there.

DIABETES INSIPIDUS.

BY T. LAUDER BRUNTON, M.D., F.R.S.

THIS disease is distinguished by great and persistent increase in the quantity of urine, with absence of sugar or albumen. It is generally accompanied by thirst, and sometimes causes wasting of the body.

HISTORY.—The disease termed diabetes by Galen and Aretæus includes cases of both diabetes insipidus and mellitus, for until Th. Willis suspected the presence of sugar in the urine in some cases and not in others, it was impossible to distinguish between them. Succeeding authors, however, left this ground of distinction almost unrecognised. About the middle of the eighteenth century, Sauvages distinguished diabetes mellitus from other diseases in which the amount of urine was greatly increased. Cullen observed that in some instances the urine was not sweet, and Franke distinguished between the two sets of cases by giving to the one the name of diabetes mellitus, and to the other that of diabetes insipidus.

ETIOLOGY. *Sex.*—Like diabetes mellitus, this disease occurs more frequently amongst men than women—the proportion being three men to one woman in both diseases.

Age.—It occurs at all ages, but seems, like diabetes mellitus, to be most common in adults. The period of maximum frequency, however, appears to be earlier than in the other disease, for Roberts, Strauss, and Van-der-Heijden, all agree in placing its maximum of frequency in the decade between twenty and thirty years of age.

	Roberts.	Strauss.	Lacombe.	Van-der-Heijden.
Infancy	7	9	—	2
From 5 to 10 years . . .	15	12	2	5
,, 10 to 20 ,, ...	13	—	7	19
,, 20 to 30 ,, ...	16	57	5	23
,, 30 to 40 ,, ...	—	—	6	19
,, 40 to 50 ,, ...	15	—	—	9
,, 50 to 60 ,, ...	—	7	5	6
,, 60 to 70 ,, ...	4	—	1	4
Above 70	—	—	—	—
	70	85	26	87

A certain tendency to heredity appears to exist, and persons of a nervous temperament seem more predisposed than others to this disease.

Exciting Causes.—The chief exciting causes seem to be injury to the nervous system, such as blows on the head, tumours in the brain, hæmorrhage, inflammation, and degeneration of the brain substance, sudden or powerful emotion, spinal meningitis, or such conditions as greatly increase the secretion of urine normally, *e.g.* exposure to cold, which by checking the perspiration throws more work on the kidneys, the excessive use of spirits, large draughts of cold fluids, &c. Great physical exertion or violent muscular efforts have also occasionally been followed by the occurrence of this disease. It has also been observed to come on during recovery from fevers, either continued or remittent. Excessive variation of temperature seems sometimes to have occasioned it, and in other instances it has been observed as a consequence of privation.

Symptoms.—The chief symptoms of this disease are, the excessive quantity of urine secreted, and the consequences which this loss of water produces. These are thirst and diminished secretion from the other glands, such as the sweat and salivary glands, causing dryness of the skin, mouth, and fauces. Sometimes, instead of dryness of the mouth, there is ptyalism. As in diabetes mellitus, the skin, instead of being dry, may sometimes be normal, or subject to profuse perspiration. Boils, which so commonly occur in saccharine diabetes, rarely appear in diabetes insipidus, but the face is sometimes liable to erythematous congestion. The general health is often good. The appetite is usually moderate, though sometimes it may be excessive. The urine is passed in enormous quantities, even greater than in diabetes mellitus, and not unfrequently averages from fifteen to forty pints daily. Its quantity is greater than that passed by healthy persons drinking the same amount of fluids, probably because less passes off by the skin. There is also a difference in the effect of fluids upon the urinary excretion. When a healthy individual drinks a quantity of fluid, the quantity of urine becomes increased shortly afterwards, but the increase does not last long, whereas in patients suffering from the complaint now under consideration, the increase in the quantity of urine after the ingestion of fluids takes place slowly, but lasts a long time. The specific gravity is very low, sometimes being little more than that of pure water. On an average it is from 1,003 to 1,007. The urine is very pale, almost like water, its reaction is slightly acid, but it is more prone to alkaline decomposition than ordinary urine, and quickly becomes neutral or alkaline. The proportion of solids in it is greatly under the normal, but the total of the solids contained in the urine passed by the patient in twenty-four hours usually exceeds the normal quantity.

Robert Willis in 1830 distinguished three sorts of diabetes, according as the urea was increased, normal, or diminished. The first he called azoturia; the second, polyuria; the third, hydruria. The solids in the urine, however, coincide so closely with the amount of food ingested, that all these varieties might be produced in a single individual by alterations of the diet. Usually there is an increase in the amount of solids as well as in the water of the urine. The urea, sulphates, and phosphates are all increased in much the same proportion. Uric acid and creatinine are said to be diminished; hippuric acid has been observed in one case, and inosit has appeared in several, but inosit occurs in the urine of healthy persons who drink much water, and even, it is said, in larger quantity than in the urine of cases of diabetes insipidus.

Course and Duration.—This disease generally begins gradually and the secretion of urine, as well as the thirst which the loss of water from the body occasions, may continue for some time before they attract the attention of the patient. In other cases they may be preceded by prodromata, indicative of nervous disturbance, such as sleeplessness, headache, irritability, cramps, partial aphasia, and disturbances of sensibility and motion in the extremities. At other times the disease, like diabetes mellitus, may begin suddenly, the patient's attention being attracted by the unusual quantity of urine he passes, or by his thirst.

A sudden onset has been observed during recovery from febrile disease, after injury to the head, exposure to cold, a fit of intemperance, violent muscular effort, and sometimes without any apparent cause whatever.

Except where it depends upon, or where it has been caused by some traumatic injury, its course is generally slow, and in one instance recorded by Willis, the patient affected lived for fifty years. Sometimes it seems to undergo intermission. The thirst and polyuria continue for some time, and then diminish, again reappearing with their former intensity. The disease is aggravated by violent emotions. Unlike diabetes mellitus, it seems to be very little influenced by diet. Inter-current attacks of fever diminish both the thirst and the urine, at the same time that they increase the specific gravity of the latter. After these attacks have passed off the patient may either return to his former condition, or a permanent improvement may continue.

Excepting for the tormenting thirst and the annoyance caused by the frequent calls to micturition, the patient's health is often very little affected.

The duration of the disease varies much. It has caused death in as short a space as four months, but it usually lasts for many years. Sometimes emaciation sets in, the appetite fails, and the patient dies of exhaustion, but death generally occurs from some complication, such as inflammation of the lungs, or disease of the brain or cord. In the latter case, there may be affections of general sensibility and touch, hyperæsthesia or anæsthesia, headache, which may be either constant or intermittent, affecting the forehead or sinciput, trembling of the muscles, convulsive twitchings, and coma preceding death.

PATHOLOGICAL ANATOMY.—Of the conditions found after death in this disease the most constant seem to be alterations in the structure of the kidneys and brain. In three cases out of nine Roberts found atrophy and degeneration of the renal substance, in a fourth the glandular tissue of the kidney was entirely wanting, in a fifth multiple abscesses were present in these organs, in two others the kidneys were hyperæmic and somewhat enlarged, in two others fatty degeneration of the nervous tissue was found in the walls of the fourth ventricle, and in one a tumour was present in the brain. Other authors have observed inflammatory and degenerative changes in the medulla oblongata and brain, tubercular or sarcomatous tumours involving these parts of the nervous system, syphilitic exostoses on the cranial bones, enlargement of the mesenteric glands and degeneration of the solar plexus.

PATHOLOGY.—The researches of Claude Bernard and others have shown that diabetes mellitus, in many instances at least, probably depends on dilatation of the hepatic vessels and a freer flow of blood than normal through the liver. The pathology of diabetes insipidus appears to be somewhat similar, only that the renal vessels are dilated instead of the hepatic ones. The diameter of the blood vessels, both of the kidneys and the liver, is regulated by the vaso-motor centre in the medulla oblongata. The nerves of the kidney pass down through the spinal cord and the splanchnics, and along the renal artery to the hilus. When they are divided as they pass along the renal artery, the flow of blood through the kidney is increased, the pressure in the glomeruli becomes greater, and the urine is secreted more abundantly. When the spinal cord, or the splanchnics themselves are cut across, the secretion is diminished instead of increased, because the vaso-motor nerves of the intestines are divided as well as those of the kidney, and just as we have seen in the case of the liver, the secretion through the kidneys is diminished by the blood going to these other vascular districts instead of flowing through the renal vessels. Like the hepatic vessels also, the renal arteries may be dilated reflexly by puncture of the floor of the fourth ventricle, by irritation of the central end of the vagus, or by injury to certain parts of the cerebellum.

Not only can the profuse secretion of urine in diabetes insipidus be explained by the supposition that in it the renal arteries are in a state of permanent dilatation, but this hypothesis also affords an explanation of the difference between the effect of fluids on the urinary secretion in patients suffering from this disease and healthy persons. In the latter the ingestion of a quantity of fluid seems to cause increased determination of blood to the kidney and rapid secretion of urine. The excess of water is thus quickly removed from the blood, and the urine soon falls to its normal standard. In cases of diabetes insipidus the renal arteries are already fully dilated, and any addition of water to the blood consequently has but slight power to increase the renal circulation. The urinary secretion is therefore not increased in the same way as normally, but the increase, although slight, goes on for a longer time, until all the excess of fluid has been removed from the blood. Some authors have been disposed to look upon the thirst and polydypsia as the primary condition, and the polyuria as merely consequent upon them. This view, however, is probably incorrect, and treatment based upon it has proved unsuccessful. When the quantity of liquid allowed to patients suffering from diabetes insipidus has been limited, the excessive thirst has induced them even to drink their own urine, and when the supply of drink has been completely cut off, the secretion of urine appears to go on at the expense of the tissues, the patient becoming thinner on account of loss of water from the body. An examination of the blood in this disease has shown it to be comparatively poor in water, and rich in solids. These conditions point to the polyuria as the cause of polydypsia. It has already been mentioned, in the article on diabetes mellitus, that if the kidneys be artificially stimulated to secrete urine, thirst is produced.

The diminution in the sweat and salivary secretion, and the consequent dryness of the skin and mouth, are probably simply results of the abstraction of water from the body by the kidneys.

The salivation which has sometimes been observed in diabetes insipidus has been attributed to irritation of the nerve centres, for Eckhardt, Nöllner, Külz and Grützner have found that puncture of the floor of the fourth ventricle close to the point which causes polyuria will produce ptyalism in dogs and rabbits. The increased amount of urea depends chiefly on the quantity of nitrogenous food eaten, for in a case recorded by Dickinson the urea varied from six to twenty-four grammes, according to the nature of the diet.

If more butcher meat than the average be taken the amount of the urea excreted will certainly also rise above the average. Similarly, diminution in the quantity of nitrogenous food will undoubtedly produce a corresponding diminution in the quantity of urea.

The ingestion of such a large quantity of water may also be to some extent the cause of the increase in the urea, for it produces this effect even in healthy persons.

DIAGNOSIS.—It is well known that the quantity of urine may be temporarily very much increased above the normal by many conditions, such as anxiety or fear; arrested secretion of the kidneys, as, for example, by sudden exposure to the cold, especially if the patient has been accustomed to warmth for some little time, or the ingestion of a large quantity of fluid such as will occur after indulgence in highly spiced and salt provisions. In hysteria also, the patient is liable to secrete enormous quantities of urine. This only takes place after a fit, and is not persistent while the polyuria in diabetes insipidus is permanent. The most important condition with which this disease is likely to be confounded is granular kidney. From this it must be distinguished by the absence of albumen in diabetes insipidus, and its presence, though sometimes only in very small quantity, in cases of granular kidney.

PROGNOSIS.—This disease resists treatment, and is rarely cured. The duration of life, in general, does not seem to be greatly shortened by it. Trousseau, however, regarded it as a more serious disease than diabetes mellitus, and as running a somewhat similar course. The cases observed by him appear to have been those in which the appetite was greatly increased, and he states that the bulimia is succeeded by anorexia, diarrhœa, and wasting, the skin becoming withered, the breath fœtid, and sometimes by phthisis.

It would thus seem that excessive appetite is an unfavourable symptom in this disease, while a normal appetite is prognostically favourable.

TREATMENT.—Numerous remedies have been employed in this disease, but with comparatively little effect. Acting on the notion that the polyuria was a consequence of polydypsia, patients have been put on a limited allowance of fluid, but without any good result, the tormenting thirst becoming increased, and the general condition worse. Diuretics, such as nitrate of potash and cream of tartar, have also been used, and the former was lauded by Franke as a certain remedy. Subsequent observations, however, have not substantiated this opinion. Nervous tonics and sedatives have both been employed. Opium at once suggests itself as a most likely remedy, from its great efficacy in diabetes mellitus, but the hopes entertained regarding its utility have not been fulfilled. In some instances it renders the patient worse. Camphor, valerian, and valerianate of zinc have all been found useful, valerian especially being recommended by Trousseau as the best remedy. Iron, ergot, strychnia, turpentine, balsam of Tolu, copaiva and acetate of lead, have all been tried with more or less success.

In cases where the disease probably depended upon some lesion of the nervous system of syphilitic origin, mercury and iodide of potassium have proved efficacious. Acting on the hint afforded by the temporary suspension of thirst and diuresis during an inter-current febrile attack, Roberts recommends the application of a large blister to the nape of the neck or the epigastrium. Galvanism has been found useful. It may be applied either by placing one pole on the nape of the neck and the other on the loins or epigastrium, or by one pole over the loins and the other pressed deeply into the corresponding hypochondrium, first on one side and then on the other

§ VI. DISEASES OF THE URINARY ORGANS.

A. KIDNEYS.
    NEPHRALGIA.
    DISEASES OF THE RENAL BLOOD-VESSELS.
    HÆMATURIA.
    ENDEMIC HÆMATURIA.
    HÆMATINURIA AND PAROXYSMAL HÆMATINURIA.
    ALBUMINURIA.
    CONGESTION.
    BRIGHT'S DISEASE.
    CONSECUTIVE NEPHRITIS AND PYELITIS.
    CALCULOUS DISEASE.
    HYDRONEPHROSIS.
    ABSCESS.
    GROWTHS.
    ANOMALIES OF FORM AND POSITION.

B. URETERS.

C. BLADDER.

NEPHRALGIA.

By W. R. Basham, M.D., F.R.C.P.

Derivation.—*νεφρὸς*, the reins or kidneys; *αλγος*, suffering or pain; hence *νεφραλγια*, pain in the kidney.

Definition.—Pain, uneasiness, or discomfort in the lumbar region, extending from one or other kidney through the ureter to the neck of the bladder and extremity of the urethra; accompanied by great and often distressing frequency of micturition; without febrile disturbance or other symptoms of inflammatory action, but with evidence of sympathetic irritation in parts connected with the kidneys through the solar, renal, intestinal, and spermatic ganglia, as shown by nausea, vomiting, pain in the testicle, and numbness more or less marked in the outer cutaneous surface of the thigh, through the connection of the external crural cutaneous nerve with the ganglia above mentioned. The urine is sometimes scanty, more often abundant, highly acid, with or without sedimentitious deposits, but frequently with crystalline or amorphous sediment—lithic acid or lithates of ammonia or soda—(red sand or gravel), and frequently exhibiting in the field of the microscope oxalate of lime crystals (*Oxaluria*); mucous or exudation corpuscles are more abundant than in health, and form a flocculent cloud when the urine has been set at rest for an hour or two.

Nephralgia is symptomatic of lithæmia, lithiasis (lithuria), oxaluria, calculous disease of the kidney, malignant and tubercular disease of this organ.

Ætiology.—The most common cause of nephralgia is lithæmia, a redundancy of nitrogenous excrementitious matter in the shape of lithic acid, retained in the organism. It may accumulate from imperfect oxidation and conversion into the more soluble and more easily excreted urea: or it may be formed largely in excess of what the organs are capable of excreting; or, as happens sometimes, constitutional idiosyncrasy or hereditary causes may, in spite of all care and management, develop a tendency to this state of lithiasis, just as gout will show itself hereditarily in opposition to every effort to control it. Redundancy of lithic acid will show itself in various ways. Thus it may appear as crystalline grains deposited from the urine as it cools, constituting red sand, often likened to cayenne pepper grains ; these are for the most part rhombic, lozenge, barrel, stellar-shaped crystals of lithic acid. Or the sediment may be of a brick-dust red (lateritious), non-crystalline in the mass, but presenting many octohedra of oxalate of lime among the granular masses of lithates of ammonia and soda of which the sediment is largely composed. The red colour is due to some molecular change which the lithates have undergone, by which purpurine-urerythrine has been abundantly formed, a condition of the urinary sediment always connected with hepatic and gastric derangement. The appearance of this sediment in the urine in cases of nephralgia is always favourable to the ready subsidence of the symptoms. But the lithic acid may not be so readily excreted. It may be so much in excess as to lead to the retention in the tubules of the kidney, either of crystalline grains or of deposits of non-crystalline lithates, which, while lodged within the tubular structure, will excite continuous irritation, even to the extent of simulating symptoms of calculus of the kidney. Such crystalline deposits in the tubules of the kidney have occasionally been demonstrated in post-mortem examinations. These concretionary masses are, after a time, washed out of the large straight tubes by the urinary current, and will pass down the ureters into the bladder, being voided through the urethra as sand or gravel, and that may be collected in the urinary sediment. These conditions give rise to the more serious and persistent symptoms of nephralgia, and should any portion of these masses not freely escape, but be detained in its original position in the tubular structure, or in its passage become lodged in any of the cup-shaped expansions of the pelvis of the kidney embracing the pyramids, slowly but continually, it will increase in bulk, and eventually develop the characteristic symptoms of renal calculus or of calculous pyelitis, often preceded by severe nephralgia and renal colic.

Nephralgia is also occasionally met with in tubercle of the kidney as well as in malignant diseases of those organs.

SYMPTOMS.—It is seen from what has been stated above that nephralgia is not symptomatic of one, but of several disorders of the urine and urinary apparatus ; and that various forms of renal disorder, both organic and functional, may develop the group of symptoms which has been embodied in its definition. It becomes therefore of practical value to establish a differential diagnosis between the several affections which have such a group of symptoms in common. Nephralgia may be intermittent and paroxysmal, or it may be persistent and continuous. In the first division it is the usual concomitant of lithiasis, whether in the form of urine highly charged with lithic acid, soluble while passing through the passage, but forming a crystalline deposit after the urine has cooled; or in the other form where the lithic acid exists in the form of sand or gravel, its insolubility, while within the organism, being dependent on its proportion, this being in excess of what the acid phosphate of soda can hold in solution. In this it is therefore symptomatic of functional disturbance, and holds out the prospect of being amenable to treatment.

In the second division, the symptoms being more or less persistent, it is usually attended by conditions, both of the urine and of the general health, which indicate a disorder more decidedly organic, and affecting the integrity of the renal organs themselves.

The simplest and most amenable form of nephralgia is that excited by the lowest form of lithiasis—that in which a more or less copious deposit of red crystalline grains of lithic acid is formed on cooling. A certain amount of lumbar pain, an aching uneasy sense of weight, may at random be called nephralgia; but unless the pain or uneasiness be accompanied by some special conditions to be presently described, the term would be misapplied—thus lumbago has been often mistaken for nephralgia.

The character of the pain in nephralgia is almost as various as the patients who suffer—or, in other words, hardly any two patients describe their sensations alike. Sometimes the pain is shifting and irregular, extending to the crest of the ilium, and then downward, following the course of the ureter, on one side or the other, with a sense of aching or uneasiness referred to the extremity of the urethra, with a frequent desire to pass water, which is accompanied by a sense of heat or even scalding in the urethral passage. This more frequently occurs in women. Other sensations are described by patients having much the character of rheumatism, the large cushion of the gluteal muscles being their seat. Uneasy sensations referred to the muscles of the thigh, with a sense of numbness in the cutaneous surface, are not infrequent. These symptoms are invariably accompanied by increased frequency of micturition, which at once gives the true interpretation to their origin, and refers them to sympathetic irritation derived from the renal organs, and not originating as rheumatic pains in the several muscles themselves.

The urine in many cases has no morbid character visible to ordinary observation, but when chemically and microscopically examined, lithic acid is found largely in excess of the healthy average. The sediment often exhibits red crystalline grains, and, adherent to the inner glazed surface of the chamber vessel, bright orange-red grains of lithic acid are precipitated. When such urine is allowed to cool in a glass vessel these red crystalline grains of lithic acid are seen in abundance adherent to its sides. Lithic acid may be thrown down in still greater excess, forming rapidly a perfect crystalline precipitate of a yellowish red sand, which microscopically consists of the many forms which lithic acid assumes in its crystalline shapes.

The nephralgia co-existing with this abundant excretion of lithic acid is rarely accompanied with any great disturbance of the general health. It may cease spontaneously; or it may cease in a few days under judicious treatment, and may return as the result of some dietetic imprudence. It may thus return at intervals for years, or it may eventually, particularly by careful diet, disappear altogether, but the majority of individuals are careless of the future, and unmindful that these attacks are the premonitions of the gouty diathesis, and are but too frequently the precursors of a genuine attack of gravel.

The nephralgia co-existent with the passage of gravel differs in no respect except in degree from that which is developed by lithic acid in excess. The most uniform diagnostic group of symptoms consists in a constant irrepressible desire to pass water, although but a small quantity may pass, attended, or it may be more properly said, preceded, by a darting pungent pain at the neck of the bladder and extremity of the meatus urinarius—which is momentarily relieved by the act of micturition—to return again and again after shorter or longer intervals, relieved each time by the passage of the small quantity of urine present in the bladder, and returning so soon as a very small quantity has accumulated. Each act of micturition is accompanied by a burning, cutting pain along the urethra, as if the passage were denuded of its protective epithelium. In mild cases the urine is not at first turbid; on settling in a glass vessel a coarse sediment falls, of a fawn colour, and composed of particles like coarse sand or even gravel. This sediment is partly crystalline and partly non-crystalline. The first shows many of the crystalline forms of lithic acid. The latter presents an amorphous appearance, granular, with minute globular particles, which further analysis proves to consist of lithates of ammonia and soda.

If the attack of gravel be prolonged, and the particles acquire a coarse and gritty character, isolated blood-discs may be seen in the field of the microscope, proclaiming the presence of blood in minute quantity, and even heralding an attack of hæmaturia. In these cases the appearance of the crystalline forms of lithic acid are particularly suggestive of the possible occurrence of hæmorrhage as the result of local irritation. They are distinctly stellar, made up of acicular crystals conjoined in their centre, and it is very easy to conceive how much more pungent and painful must be the passage of such crystalline masses over the surfaces of the tubes and outlets of the kidneys. The urine in such cases is generally of average specific gravity, and occasionally contains a small amount of albumen, which is usually only proportionate to the number of blood discs present. In old gouty cases the larger proportion of albumen must excite attention to the probability of the existence of a gouty contracting kidney.

Nephralgia symptomatic of the more serious affections associated with the renal apparatus, namely, calculous, malignant, and tubercular disease, is accompanied with the other clinical phenomena which are characteristic of each of these complaints, and these are described in separate articles.

Treatment.—A paroxysm of nephralgia with its urgent and distressing symptoms, if arising from a lithuria or lithiasis, that is, from an abundant excretion of lithic acid, and the passage through the renal organs of a highly acid urine, or of urine surcharged with crystalline grains of lithic acid, is usually very amenable to judicious treatment. Two therapeutic objects must be kept in view:—

Firstly, the neutralization of the highly acid and irritating urine.

Secondly, the use of remedies to excite the sluggish and imperfect hepatic function, which must be regarded as the remote cause of the urinary disturbance.

To attain the first object without considering the necessity of the second would be only temporarily, and for a very short period, to afford relief to the patient. Keeping both objects simultaneously in view, complete relief is quickly obtained.

According to habit or idiosyncrasy of the patient, the treatment should begin with either a brisk mercurial purge, or, what is better, with small alternate doses of blue pill and rhubarb, to be followed each morning with some neutral saline. The Carlsbad, Püllna, or Friedrichshall waters are most efficacious. Moderate quantities—a few ounces—should be taken warm, either every morning or on alternate mornings, while the patient is fasting. The bicarbonate of potash, with or without some salt of lithia, should be taken twice or three times a day in an effervescing form with lemon-juice. To render these measures more promptly remedial, a strict attention to diet should be enjoined. Animal food should be restricted; and wine, beer, and other stimulants given up, or if that be not done, then their quantity, quality, and the period of the day when they are taken must be strictly regulated.

In regard to stimulants it may be said of them all that the mischief they are capable of producing is largely proportional not only to the quantity taken, but to the time when taken. On an empty stomach they produce the maximum amount of injury; taken with food the evil is then proportional to the quantity and kind. Taken on an empty stomach the injury is twofold:—1. On the gastric glands and mucous surface of the organ, by which the primary stage of gastric digestion (when subsequently called into action) is weakened and damaged. 2. The alcoholic fluid passes undiluted or unmodified into the portal system and hence disturbs and deranges the important function of the liver; and it may lead eventually to disorganization, obstructed circulation through the organ, ascites, hepatic dropsy, and death.

In the treatment of simple nephralgia, those stimulants possessing a limited action are preferable to those more distinctly acting as alcoholic excitants. All are better avoided; but it so frequently happens that those suffering from nephralgia have become habituated to their use, that it is not advisable, indeed it is impracticable, to forbid their use altogether. Selection should therefore be made of those which are best adapted to the remedial agency required. In addition to the main object of treatment—the cutting off the formation of lithic acid in excess—must be the evacuation from the renal organs of any portion already formed and detained within the renal tubes. This point is attained by the use of those stimulants only which possess a diuretic action. Gin, hollands, or whisky in moderate quantities, largely diluted, will keep the renal tubes well washed by the copious stream of a diluted urine, which effectually carries away any irritating grains of lithic acid lodged in them. The stimulants to be avoided are all the strong and heady wines—port, sherry, and madeira; all effervescing wines, especially the sweeter or more fruity sorts of champagne; all the sparkling varieties of hock or burgundy; all kinds of beer, whether in draught or bottle; all liqueurs. On the other hand, the wines best suited for these disorders are all the light kinds of claret, or various kinds of hock diluted with seltzer-water.

The amount of animal food should be reduced and limited to one meal daily, and the diet otherwise regulated according to the character of the dyspeptic symptoms experienced by the patient.

CONNECTION OF LITHÆMIA, LITHIASIS, OXALURIA, AND HYPOCHONDRIASIS.

Nephralgia arising from lithiasis is so intimately connected with symptoms of other functional disorders, that the subject would be incomplete without a review of their most prominent and characteristic features. These are chiefly referable to the digestive process, either in the primary or secondary stages; and to the influence of such imperfect processes on the nervous and circulating systems.

They may be enumerated as inappetency; flatulence and uneasy sensations after food; acid eructations and heartburn; torpid bowels; defective taste; mental depression or hypochondriasis; and intermittent or irregular pulse. In these cases the nephralgia is by no means a leading symptom. There may be some frequency in micturition, the urine passed being small in quantity, highly acid, and containing lithic acid crystals, deposited on cooling. This sediment, when examined with a microscope, is seen to consist of crystalline grains of lithic acid, mixed with a large proportion of octohedral crystals of oxalate of lime. In many cases the sediment consists entirely of these octohedra.

The co-existence of this form of sediment with the symptoms of gastric disturbance, nervous and mental depression (hypochondriasis), and irregular and intermittent pulse, induced some writers on urinary pathology, notably Prout and Golding-Bird, to refer these several disturbances in the gastric assimilation, nervous, and circulating functions, to one common cause, under the definite term of *Oxaluria*, or the *Oxalic acid diathesis*.

Dr. Prout,[1] who first described the symptoms of this diathesis, believed them to belong to the irritable or nervous class, rather than to the congestive or inflammatory. He stated that they were marked by irregularity of the heart's action, intermission of the pulse, palpitation, flatulent disorder of the stomach, with more or less hypochondriasis, depression, and low spirits.

Dr. Golding-Bird endorsed these views, and added, by his popular work on urinary deposits, still further to the belief and recognition of the diathesis under the term, first employed by him, of *Oxaluria*. There were, however, not wanting physicians and chemists who expressed themselves as sceptical of this oxalic diathesis. Among the earliest writers who entertained doubts of this oxaluric theory was Dr. Owen Rees. In his work, published in 1845, "On the Analysis of Blood and Urine in Health and Disease," he observes:—"The state of the system on which the secretion of urine characterised by deposits of oxalate of lime depends is not well investigated. There appears some degree of probability that it is connected with the formation of lithic acid in excess, and with a state of system in which a considerable quantity of urea is secreted." Still later, in the Croonian Lectures for 1856 at the College of Physicians, Dr. Owen Rees affirmed that oxaluria was not indicative of a diathesis, but that the oxalate of lime was formed after the urine had been secreted by the kidneys, and was derived from the uric acid and urates of the secretion. It is remarkable how long an erroneous theory will prevail when originating with observers of reputation. There are many who yet view oxaluria as a distinct disorder, and who consider it to be something entirely different from the lithic acid diathesis, and continue to accept the view of Golding-Bird that the irritable form of dyspepsia, with nervous depression and hypochondriasis, already mentioned, are symptoms peculiar to the presence of oxalate of lime, rather than common to a state of lithæmia with corresponding excess of lithic acid in the urine. To prove that lithic acid is the only source and origin of oxalic acid and the oxalates in the urine, it will be necessary to trace the formation of oxalic acid, and to show, first, that perfectly fresh urine presents no trace of oxalate of lime crystals, and that it is only after a short exposure to the air that they become visible in the sediment.[1]

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[1] Stomach and Renal Disease, p. 62 et seq.

In tracing the source of oxalic acid in the urine, the ready formation of it by the oxidation of most organic compounds must not be disregarded. It is abundantly formed by the oxidation of sugar by nitric acid, also by the decomposition of the urate of ammonia, whether obtained from human urine or from the same compound contained in guano.[2] The chemical relations of oxalic acid to lithic acid and the lithates have been investigated by chemists of the highest reputation, and all concur that the oxalic acid is one of the products of the oxidation of these organic excretions. The following facts sufficiently prove this proposition:—

Lithic acid is converted into oxalic acid and urea by the addition of oxygen and water.

Lithic acid, mixed with a fermenting agent (yeast) and an alkali, with an elevation of temperature, is decomposed into oxalic acid and urea.

Lithic acid, heated with water and peroxide of lead, is converted into urea, allantoin, oxalic acid, and carbonic acid (Wohler and Liebig).

Lithic acid, digested in chlorine water, forms alloxantin, alloxan, parabasic acid, and oxalic acid.[3] Lithic acid oxidised by nitric acid yields a large quantity of parabasic acid, oxalic acid, and ammoniacal salts.[4]

Examples might be multiplied to prove that the oxidation of lithic acid always results in the formation of oxalic acid, urea, and other compounds. The lithates undergo similar decompositions.

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[1] Lehmann, Physiological Chemistry, vol. i. p. 44.

[2] Gmelin, Handbook of Chemistry, vol. ix. pp. 111 et seq. Cavendish Soc. Edition.

[3] Ibid. vol. x. p. 460.

[4] Ibid. p. 462.

It is well known that Peruvian guano, which is almost pure lithate of ammonia, becomes converted in the hold of the ship very largely into oxalate of ammonia. These facts not only support, but prove the opinion expressed so many years since by Dr. Owen Rees, that the oxalate of lime is formed after the urine has been excreted by the kidneys, and is derived solely and directly from the decomposition of lithic acid and the lithates. But the subject has been still further and successfully investigated by Edward Schunck, F.R.S., in a paper published in the Proceedings of the Royal Society, "On Oxalurate of Ammonia in the Urine."[1]

The process for obtaining the oxalurate is given in detail. Whether the oxaluric acid exists originally in a free or combined state is a matter of doubt, for it is quite possible that the ammonia may be derived from the decomposition of the urea present in sufficient quantity to saturate the oxaluric acid present. With regard to the origin and source of the oxaluric acid, Mr. Schunck distinctly states that there can be little doubt that in the animal frame, as in the laboratory, it is formed by the oxidation of lithic acid, which is its only source. This conversion of oxaluric acid into oxalic acid and urea may take place in any part of the urinary apparatus, or only in the urine after excretion and exposure to the air. Few organic compounds are so unstable or so readily undergo decomposition as the oxamic series. Acids, alkalies, or even water at a certain temperature, will decompose oxaluric acid. Urine containing oxaluric acid will, by simple exposure to the air, yield oxalic acid and urea, and consequently crystals of oxalate of lime. In the majority of cases perfectly fresh urine contains no oxalate of lime,[2] while the same urine, after a short exposure to the air, exhibits abundant evidence thereof. The same urine boiled exhibits similar results.

The decomposition of oxaluric acid may take place in any part of the urinary apparatus after the urine has been once secreted; and hence an intelligible explanation is offered of the formation of oxalate of lime, whether in the urine only and subsequent to exposure to the air, or even within the renal tubes, as must happen in cases where oxalate of lime calculi are formed, or even in lithic acid calculi, the nucleus of which is so frequently found to be composed of this oxalate of lime salt.

The oxaluric acid, on its combination with ammonia, rapidly undergoes decomposition when brought into contact with either acid or alkaline salts, and therefore will, in the presence of the acid phosphates, or even of the basic phosphates of soda in the urine, become converted into oxalic acid and urea, and the former, with its powerful affinity for lime, would, at the moment of formation, unite with or decompose the lime salts always present in the urine. Mr. Schunck very justly concludes that this oxalurate ($C_5N_2H_8O_8$) may be considered the vehicle by which the oxalic acid escapes from the system in the least injurious form.

Such are the leading and most characteristic chemical relations of oxalic acid to urea, lithic acid, and the lithates, and in the face of these chemical facts it must be admitted that the opinion expressed by Dr. Owen Rees, that the probable derivation of oxalic acid and its oxalates from lithic acid, is fully established and confirmed.

All urines, therefore, whether from persons in health or suffering from diseases of the most varying character, may at times contain oxalate of lime crystals. They are, consequently, not significant of nervous depression or hypochondriasis, or of irregular or intermittent pulse, or of any special form of dyspepsia, any more than they are significant of emphysema, chronic bronchitis, phthisis, or even diabetes, in all of which disorders an abundant sediment of oxalate of lime is occasionally found.[1]

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[1] Proceedings of the Royal Society, vol. xii. No. 95.

[2] Lehmann, Physiological Chemistry.

The chemical facts already stated may, it is hoped, dissipate that chaos of opinion which the late Dr. Parkes so emphatically stated to exist on this subject.

Oxalate of lime as a sediment in the urine is of no other significance than that of a lithic acid diathesis (lithiasis). When seen in urine immediately on being passed, and before such urine has had time to cool, it may signify that the oxalurate of ammonia has undergone molecular change within the urinary or renal passages, and the formation of an oxalate of lime calculus is thus considered possible.

The symptoms then of the disorder which Dr. Golding-Bird designated as oxaluria are closely associated with, if not entirely dependent on, a state of the organism which Dr. Murchison has very appropriately termed lithæmia,[2] a condition in which oxidation is imperfectly performed, and insoluble combinations of lithic acid and lithates, instead of the readily soluble urea, are the results. This state of lithæmia, with its concomitant condition of lithiasis, and deposit of oxalate of lime crystals, exhibits all the characteristic and typical symptoms of the oxaluria of Golding-Bird, namely: More or less frequency of micturition, occasional sense of heat, or even scalding, along the urethral passage, dyspeptic symptoms of varying character, flatulent disturbance after food, irregular and frequently intermittent pulse, with occasional attacks of palpitation, sluggish torpid bowels, depression of spirits assuming the character of hypochondriasis, restless nights and unrefreshing sleep, with its attendant feeling of weariness and languor. These symptoms are most commonly met with in persons of indolent habits, in dyspeptics, whose errors of diet and regimen lead prominently to defective liver formation, and they are not rarely seen in those whose occupations, literary or professional, subject them to great mental strain, and who, from prolonged mental occupation, are deprived of the exhilarating influence of air and exercise. Hypochondriasis in these cases is frequently accompanied by various delusions, of which the most common relates to the sexual functions, a dread of impotency becoming the predominating thought; or if this be not the form, then some subjective symptom occupies the mind, and the enjoyment of life is subordinate to its influence.

TREATMENT.—In considering the treatment of this group of functional disturbances, it is most essential to keep prominently in view the pathological fact that it is not the kidneys that are at fault, whose office it is to excrete from the organisms such products of the disintegration of the tissues as the process of oxidation has prepared for excretion, but rather those organs which minister to perfect assimilation, and are engaged in the important office of separating the excrementitious from the recrementitious.

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[1] See Parkes On the Urine, p. 221; also Roberts, Urinary and Renal Disease, p. 51.

[2] Functional Diseases of the Liver, p. 72 et seq.

The hepatic function is that to which attention should be chiefly directed. Medicinal treatment may do something, but diet and regimen can do more for the relief and cure of this class of disorders.

Whether the symptoms take the form of nephralgia, with its frequency of micturition, its lumbar pains and sympathetic sensations, its highly acid urine, with a sediment of lithic acid crystals (red sand), or are in the aggregate more allied to a wider range of disturbance, and which have been described under the name of oxaluria, in which the gastric, hepatic, nervous, and circulating systems are more obviously at fault, the ætiology of each being the same, the treatment, whether medicinal or dietetic, will not greatly differ. Looking to the fact that in this group we have to treat a lithæmia, with its accompanying lithiasis, the latter condition varying only in intensity, it may be conceded that the principles of treatment appropriate to one will be equally applicable to the other.

In the graver form of oxaluria, in which so wide a range of disturbance exists, torpid sluggish bowels, inappetency, or at least depraved gustatory sense, irregular and intermitting pulse, with hypochondriasis more or less marked, severally indicative of functional hepatic disturbance, the chief aid medicinally will be derived from alterative doses of blue pill with rhubarb, avoiding, except in special cases, the employment of active mercurial or resinous purgatives. There are no remedies which assist mild mercurial doses so efficaciously as some of the purgative bitter waters—the Friedrichshall, the Püllna, the Marienbad, the Carlsbad, or the Hunyadi Janos—all of which are most efficient as laxatives, and are most beneficent in these cases of hepatic torpor or lithæmia. They are especially beneficial when the symptoms are expressive of a gouty diathesis.

When these alterative measures have relieved the patient to the extent of obtaining improved and daily action of the bowels, the digestive powers will be materially strengthened by dilute hydrochloric acid and infusion of gentian, or, in some cases, by the nitro-muriatic acid with some vegetable bitter. These, if taken a short period before food, rapidly improve the appetite, and with an improved digestive process some of the leading symptoms will slowly subside. The mental depression gives way to a more cheerful tone; the sleep becomes sounder and more refreshing; the urine slowly loses the crystalline oxalate of lime, while the lithic acid returns to its natural proportion, and, so far as the urinary secretion is concerned, all evidence of disturbed function has disappeared. Nevertheless, among the symptoms which are typical of this form of disorder, the irregularity of the pulse, with its marked intermissions, will continue for weeks after the major symptoms have disappeared. To the hypochondriac, who habitually feels his own pulse, the continuance of this irregularity often leads to the belief in the patient that he is the subject of heart disease, and it is difficult sometimes to persuade him otherwise. The intermission is simply a pulseless systole. In ordinary cases there is no intermission of the heart's motion; all that can be detected by auscultation is a somewhat feebler impulse and a less vigorous sound corresponding to the systole. It would seem that the quality of the blood does not impart a stimulus sufficiently powerful to send the pulse-wave fully and strongly throughout the arterial system.

Irregularity of the heart's action may exist also in these cases. This symptom consists of a few beats at an ordinary rate followed by several of double or treble rapidity, now and then a marked intermission intervening. This irregularity in the pulse may, however, be indicative of organic disease, and is of graver import than an intermitting pulse. Nevertheless, the co-existence of irregularity with intermission is usually dependent on a like cause, blood surcharged with the effete material, and unequal to conveying a healthy and vigorous stimulus to the heart's action. This irregularity, therefore, is not produced by any organic defect in the heart itself. Auscultation fails to find any adequate cause for it; and the patient may have his apprehensions of organic disease of the heart allayed.

The patients in whom these suspicious heart symptoms arise are usually of robust frame and make, enjoying good health in early life, of active bodily habits, and generous livers, rather inclined to obesity than otherwise, and who, from accident or necessity, have been debarred from their usual active exercise.

The following is a typical case :—A gentleman, a landed proprietor of good estate, fifty-five years of age, following eagerly the exhilarating sports of a country life, met with a severe accident in the hunting field, a fractured thigh and humerus, and was consequently compelled to give up hunting, and subsequently also shooting. He became dyspeptic, suffering much flatulence, with sluggish bowels, occasional palpitation and fluttering in the cardiac region, with irregular and intermitting pulse. A state of hypochondriasis followed, with loss of sleep and defective appetite, and the flatulent distension after food was so great that a sense of choking, with much shortness of breath, alarmed him into the fear that he was becoming asthmatic. The urine was clear, non-albuminous, highly acid, a slight sediment showing crystalline grains of lithic acid, with an abundant crop of oxalate of lime octohedra. Constantly feeling his own pulse, he became possessed with the belief that he was the subject of heart disease, and this preyed so much upon his mind that, after sleepless nights, he became so low and depressed as to give fear to his family that his mind would give way. The intermitting and irregular pulse was most marked, sometimes an intermission every third, fifth, and tenth beat. The sounds of the heart were feeble, and now and then the systole seemed so weak as to convey the impression of a distinct pause.

This gentleman, during a period of more than twelve months, submitted to a stringent and a regulated diet, to an alterative medical treatment, chiefly by salines and alteratives, and the nitro-muriatic acid and orange-peel tincture. Daily exercise in the open air, both on foot and pony, restored the health, and all symptoms of cardiac irregularity disappeared; sleep became natural and refreshing, the appetite returned, the digestive function was performed without flatulence or uneasiness, and the mind became cheerful, active, and self-reliant. Such is the record of a typical case of lithæmia or oxaluria, with its attendant disturbances, simulating mental and cardiac disorder, but in reality caused by functional derangement of liver, and the accumulation in the blood of the products of defective oxidation and metamorphosis.

DISEASES OF THE RENAL BLOOD-VESSELS.

By Frederick T. Roberts, M.D., B.Sc., F.R.C.P.

In this article it is intended to refer briefly to the chief diseases to which the renal vessels are liable; as well as to certain conditions which affect the circulation in the kidneys. At the outset, however, attention must be drawn to the fact that anatomical peculiarities involving the renal arteries may lead to more or less serious consequences. Thus a renal artery may take an abnormal course, or there may be a supernumerary trunk, and in either case the vessel may press upon or twist round the ureter, and obstruct its channel, hydronephrosis being the result.

1. Arterial Degeneration.

The renal arteries may be the seat of atheroma or calcification, these alterations being usually associated with similar changes affecting the arterial system generally, but the frequency with which they are involved has not been satisfactorily determined. The condition may only involve more or less of the main trunk, or may extend along the smaller branches. Its chief importance depends upon the fact that it is liable to lead to obstruction of the renal circulation, either in consequence of the narrowing of the calibre of the vessels, or of the occurrence of thrombosis, this being followed by degeneration and wasting of the tissues of the kidney. It is also not uncommonly associated with the granular kidney. It is quite impossible to recognise atheroma of the renal arteries clinically. Dr. Greenfield[1] has recorded two interesting cases in which this morbid condition was present. In one, a male, aged 49, along with extreme atheroma and calcification of the aorta, the right renal artery was extremely atheromatous and diminished in its calibre, its branches being also narrowed and thickened. The corresponding kidney weighed only an ounce and a half, and was firmly adherent, granular, uneven, and wasted. The left kidney weighed ten and a half ounces, but presented a depressed area, which corresponded to complete wasting of one of its lobules, due to extensive atheromatous disease and obstruction of the arterial branch supplying it. In the second case, that of a woman aged 53, who had suffered from ovarian tumour with ascites, both kidneys were very small, but the left presented degenerative changes due to plugging by coagula of the trunk of the renal artery, and of all its branches in the hilus. The wall of the renal artery close to the aorta and for about half an inch beyond was much thickened by atheroma, and narrowed.

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[1] Pathological Transactions, vol. xxvi. p. 135 et seq.

The minute branches of the renal arteries are specially implicated in certain forms of disease to which the kidneys are liable, namely, in the granular contracted variety of Bright's disease; and in albuminoid degeneration, which commences in connexion with these vessels and the Malpighian bodies. The nature of the changes is described in the several articles which treat of these conditions.

2. ANEURISM.

The trunk of one of the renal arteries is in extremely rare instances the seat of an aneurism, and the branches may present small dilatations of a like nature. These owe their origin to previous disease of the vessel. In most cases they are only discovered at post-mortem examinations, but they may give rise to clinical signs of their existence, namely, pain referred to the region of the kidney, which may be severe; hæmaturia from time to time, should the aneurisms be small; and a pulsating tumour. An aneurism is liable to rupture, and should it be of any size, this event will probably lead to a rapidly fatal issue. When there is repeated hæmorrhage, the passage of clots may give rise to much pain, of the nature of renal colic; or they may set up pyelitis and subsequent disease of the kidney.

3. EMBOLISM AND THROMBOSIS OF THE RENAL ARTERIES—HÆMORRHAGIC INFARCTION.

The morbid changes resulting from embolism affecting the vessels of the kidney have long been observed and described, but their real nature and origin was not recognised until within a comparatively recent period, as the result of the observations of Kirkes, Virchow, Beckman, Cohnheim, and other pathologists. They are of much greater importance from a pathological than from a clinical point of view, as renal embolism is in the large majority of instances not made out during life, either because it gives rise to no obvious clinical phenomena, or because these are but secondary and subordinate to a more serious condition of the general system or of some other organ than the kidney.

ETIOLOGY AND PATHOLOGY.—Renal embolism originates in the same manner as embolism in other organs, especially the brain or spleen, namely, in consequence of particles being conveyed from the heart or great vessels, and becoming lodged in some portion of the arterial circulation of the kidney. These particles generally come from the heart, being of a fibrinous nature, and being detached either from clots in the left cavities, or from vegetations in connexion with the aortic or mitral valves, resulting from endocarditis or from chronic changes. Occasionally they consist of atheromatous material detached from the aortic valves or the aorta; still more rarely an embolon originates in a fibrinous deposit within an aneurism, as in a case reported by Dr. Murchison.[1] It may also come from other parts, and may have septic properties.

Thrombosis may result from a wound of the renal artery or of one of its branches, as in a case described by v. Recklinghausen; or from arterial degeneration. It is stated that this process may also occur spontaneously, especially in certain fevers, such as typhoid fever.

ANATOMICAL CHARACTERS.—In the main trunk or larger divisions of the renal artery, a plug, whether embolic or thrombic, may generally be recognised, but when one of the smaller branches is involved it is frequently difficult to demonstrate its presence. It is found, however, that injection driven into the trunk of the artery will not penetrate the region supplied by the obstructed vessel.

The effects of plugging in connexion with the renal arteries will depend upon the size of the vessel obstructed; and on the nature of the embolon, should the blocking be due to this cause. In the first place, the main trunk of the artery may in rare instances be obstructed, and then necrosis of the kidney may follow, as in a case described by Cohnheim and Bartels, where the left renal artery was obstructed by embolism, in consequence of which the whole of the cortical portion lost its vitality, becoming pale, dull, opaque, and dry, with scattered deep bloody red spots. This is not, however, always the consequence. Wilks and Moxon[2] mention a case where Bright's disease in both kidneys was already present, and there was a most interesting reduction of the disease in the organ the vessel of which was obstructed. Another result which has been found is the development of granular disease of the kidney. When one or more of the larger branches are blocked, intense hyperæmia of the tissue surrounding the part deprived of blood is produced, followed by rupture of the Malpighian capillaries and extravasations of blood.

The usual result of the blocking of branches of either renal artery is the development of hæmorrhagic infarctions in the kidney, for the formation of which the peculiar distribution of the renal vessels is specially favourable. These infarctions are almost entirely confined to the cortical portion, and they may be seen in different stages in the same organ. They vary considerably in number as well as in size, the latter ranging from that of a hemp-seed to that of a nut or even larger; and there may be a number of small infarctions in one section of the kidney, while the rest of the organ is free, in consequence of an embolon being broken up against the bifurcations of the artery into smaller particles, which pass into its finer divisions. A renal infarction is more or less wedge-shaped, though less distinctly so than in the case of the spleen. Its base is directed towards the surface, and projects a little beyond the surrounding level; while its apex points towards the infundibulum.

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[1] Medico-Chirurgical Transactions, vol. xlviii. p. 129.

[2] Pathological Anatomy, 2nd edit. p. 517.

Primarily renal infarctions almost always, though, it is said, not invariably, present a hyperæmic and hæmorrhagic appearance, due to intense congestion of the small vessels, with extravasation of blood into the uriniferous tubules, in consequence of some of these vessels having given way. Hence they are of a dark-red colour, and may be blackish-red, especially at the centre, where the hæmorrhage takes place, their edges being well-defined or somewhat blurred. Rarely, however, are they seen in this their original condition, having usually undergone more or less of the following alterations, which are mainly due to changes in the extravasated or stagnated blood and in the involved tissues, which undergo degeneration together; partly to the formation of inflammatory products. As the result of these processes, an infarction loses its red colour, this commencing at its centre, where it becomes gradually bright yellow or yellowish-white, while at its periphery it still presents a zone of bright redness, though by degrees the change in colour progresses to the circumference. This depends mainly on the alterations in the blood-pigment. The infarction also becomes rather larger, firmer, and toughish. Microscopic examination reveals at first unaltered renal tissue with blood-corpuscles; subsequently granular amorphous or fibrillated material, cellular inflammatory products, and altered epithelial cells, which undergo fatty degeneration; and, at a still later period disintegrated tubules. In some instances a further process of softening takes place, so that the central portion becomes a mere granular and fatty debris, or a purulent material may be formed. Usually in course of time the degenerated materials are gradually removed by absorption, while a growth of nucleated connective tissue takes place at the periphery, and thus a process of shrinking occurs, until ultimately nothing may be left but a firm, depressed, contracted, and puckered cicatrix, the renal tissue having been destroyed and removed, and its place being taken by connective tissue. It has been asserted that a cyst may result from a renal infarction, but Wilks and Moxon[1] state that they have never seen a cyst thus produced.

The time which is required for the completion of the changes just described is a matter of doubt; but it may be affirmed that their progress is somewhat chronic. In a case observed by Dr. Hermann Weber,[2] an infarction in the right kidney, supposed to be thirty-eight days old, was shrunk below the level of the surrounding tissue; others in the left kidney, which were believed to have occurred between a week and a fortnight later, were still slightly prominent, but had not the elevated red margin seen in still more recent cases. Usually, when there are a number of infarctions, they are seen in different stages of their progress.

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[1] Pathological Anatomy, 2nd edit. p. 517.
[2] Pathological Transactions, vol. xvi. p. 166.

When embolic particles lodge in the minute branches of the renal artery, so-called *capillary embolisms* are originated; but Cohnheim objects to this term, as the obstruction is always in the small arteries. They are observed in connexion with acute endocarditis, the kidneys presenting numerous minute extravasations and infarctions in its superficial part, and they may be also seen in its interior. These capillary embolisms likewise belong to one form of pyæmia, the particles being of a septic character, and setting up severe inflammatory action, ending in suppuration or even gangrene.

It is customary to find infarctions in other organs, especially the spleen, when they are present in the kidneys.

SYMPTOMS.—It only happens in very exceptional cases that renal embolism or thrombosis and their consequences can even be suspected during life, from the occurrence of any symptoms, and at the best these are of a most indefinite character; while in the large majority of cases there are no clinical phenomena whatever. Should a large embolon lodge in either kidney, this may be indicated by a sudden severe pain in the corresponding lumbar region, shooting down along the ureter towards the thigh. In a case diagnosed by Traube, the patient was wakened during the night by the occurrence of severe pain in the right loin, which was much increased by upward and inward pressure below the last rib, and also by movement of the trunk and coughing; but this soon passed away. The urine does not present any alterations, even when the trunk of one renal artery is obstructed. Should there be any cardiac disease or other condition present in which embolism is likely to happen, the occurrence of the kind of pain above described might possibly lead to the recognition of this event.

TREATMENT.—No treatment can have any direct effect upon renal embolism, but should it be suspected, the patient should be kept at rest in bed; dry heat or fomentations applied over the seat of pain; and, if this is excessive, a dose of tincture of opium or some other anodyne might be given.

4. THROMBOSIS OF THE RENAL VEINS.

ETIOLOGY AND PATHOLOGY.—The occurrence of thrombosis in connection with the renal veins may depend on *local* or *general* causes, or on the joint action of both classes; and it may arise independently, or by extension from the vena cava inferior or from the spermatic or ovarian vein on the left side. The ways in which the several causes act so as to induce thrombosis are either by compressing the renal vein or its branches, rendering the circulation feeble, or altering the constitution of the blood, so that it becomes prone to spontaneous coagulation. The chief *local* causes are:—1. Certain diseases of the kidney, namely, albuminoid degeneration, Bright's disease, and extensive cancer or other forms of tumour. 2. Severe injuries to the kidney, with consequent traumatic nephritis. 3. Neighbouring tumours or enlarged organs compressing the renal vein, such as glandular growths, a retro-peritoneal tumour, a pregnant uterus, or an ovarian tumour. The *general* causes include:—1. High fever, especially if associated with previous disease of, or injury to, the kidney.[1] 2. Pyæmia and septicæmia. 3. Marasmic and cachectic conditions, such as that resulting from prolonged diarrhœa in infants, or that associated with internal cancer. In puerperal fever the occurrence of renal thrombosis is a secondary event, following uterine phlebitis, the thrombus extending from the vena cava or ovarian vein. The left kidney is more frequently the seat of this lesion than the right.

ANATOMICAL CHARACTERS.—A recent thrombus is dark red, soft, uniform throughout, and non-adherent to the wall of the vein. Usually, however, it is observed in a more or less altered condition, having lost its colour to a variable degree, and being soft, crumbly or curdy, or else firm, organized, and adherent to or even blended with the vascular wall. The occlusion may be complete or partial, and occasionally the clot is channelled through its centre. It is variable in extent, and may reach in the one direction to the vena cava, in the other to the smallest venules that can be traced. In the latter case a section of the kidney presents a curious mottled appearance after the clots have become decolorized. The minute vessels may give way here and there, small extravasations being consequently visible. When only the trunk of the renal vein is blocked, or, it is said, even both trunks, a collateral circulation may be set up through the supra-renal veins and those of the ureters: but if the smaller branches are involved, this event cannot occur.

SYMPTOMS.—Thrombosis of the renal vein is usually not recognised during life, and under any circumstances its diagnosis is very difficult. If the obstruction is considerable, and collateral circulation cannot be established, the results to be looked for are marked diminution in the quantity of the urine, with concentration; albuminuria; hæmaturia; and the appearance of casts in the urine. Ultimately the affected kidney ceases entirely to perform its functions. There will probably be more or less constitutional disturbance.

TREATMENT.—Nothing can actually be done for renal thrombosis. It is important to recognise the conditions under which it may occur, so as to take measures for its prevention.

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[1] See cases reported by Dr. Moxon in Guy's Hospital Reports for 1868; and in Pathological Transactions, vol. xx. p. 227, and vol. xxi. p. 248.

HÆMATURIA.

By William Roberts, M.D., F.R.S., F.R.C.P.

Definition.—The term hæmaturia is employed to denote the passage of blood with the urine, whether its source be the kidneys, ureters, bladder, or urethra. It is a symptom of numerous and varied pathological conditions, of which, indeed, it is frequently the most striking phenomenon, not only to the physician, but also to the patient. The presence of blood in the urine is generally easily recognised by the peculiar appearance which it imparts to the secretion, but if the quantity be very small the microscope is required for its detection. In renal hæmaturia the urine has a dull-red smoky appearance, and deposits, on standing, a brownish sediment. When the blood is derived from the ureters, bladder, or urethra, the urine has a more distinctly sanguineous hue, and little clots are often found at the bottom of the vessel.

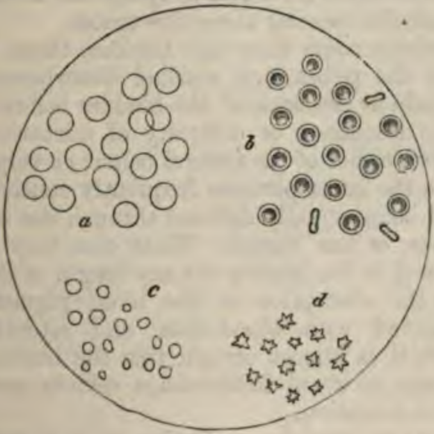
The microscope is the surest means of discovering blood in the urine. In acid urine of normal density the corpuscles preserve their characteristic forms for several days, but if the urine be very watery or ammoniacal they disappear very speedily.

The red disks do not run into rouleaus in the urine but lie freely scattered about. Their appearance under the microscope varies a good deal according to the condition of the urine. When the urine is acid and of moderate density, they exhibit more or less of the biconcave contour and deep shading which they possess when freshly drawn from the blood-vessels. (See Fig. 1 *b*.) If the urine be very dilute the corpuscles expand into plain delicate circles without any appearance of cell-contents (*a*). Sometimes their original shape is greatly changed, they appear shrivelled, indented, or crumpled, and puckered so as to be scarcely recognisable (*c*, *d*). Blood-disks in urine are apt to be confounded with torula cells, also, but more rarely, with the minute discoid forms of oxalate of lime, and with the nuclei of renal epithelium. From the first they are distinguished by the absence of a nucleus which can always be detected by a good glass in torulæ. Oxalate of lime discoids are distinguished by the existence of intermediate forms which connect them with unmistakable dumb-bells. Renal nuclei refract the light more strongly than blood-disks, and a solution of magenta gives them a deep carbuncle-red tint, very different from the pale pink hue imparted to blood-corpuscles by the same reagent.

The chief types of hæmaturia may be grouped under the following heads:—

(*a*) *Hæmaturia from local lesions.*—By far the largest number of cases of hæmaturia come under this heading; and arise under a variety of conditions, viz., acute and chronic Bright's disease, active or passive congestion of the kidneys, violent muscular effort or straining, external injury, calculus of the kidney, ureter, or bladder, abscesses of the kidney, ureter, or bladder; cancer, tubercle, or entozoa in any portion of the urinary tract; morbid conditions of the bladder or prostate (villous disease, acute cystitis, varix of vesical veins, &c.).

(*b*) *Symptomatic hæmaturia.*—In scurvy and purpura, also in certain hæmorrhagic or so-called "malignant" types of continued, eruptive, and malarial fevers the blood assumes an unnatural fluidity, and soaks out of the blood-vessels into the uriniferous canals, and imparts to the urine a dark sanguineous character. In these cases the blood-pigment escapes in much larger quantity than the corpuscles, and gives a depth of coloration quite out of proportion to the scanty numbers of blood-disks found under the microscope.



Blood-corpuscles in urine. *a.* Slightly distended by imbibition; *b.* Showing their biconcave contour; *c.* Shrivelled; *d.* Serrated.

(*c*) *Supplementary Hæmaturia.*—In some rare cases hæmaturia is distinctly vicarious or supplementary to the menstrual discharge or a hæmorrhoidal flux or a paroxysm of spasmodic asthma.

In a case of hæmaturia it is of the first importance to determine the source of the blood, that is to say, to discover whether it comes from the kidneys, the ureters, the bladder, or the urethra.

In determining whether the blood comes from the kidneys or from the bladder, we are guided partly by subjective, and partly by objective phenomena. Thus, if the kidneys are the source of the bleeding, in most cases there will be symptoms referrible to those organs, viz., heat, weight, or pain on pressure in the lumbar region. There may also be pain along the course of the ureters, extending along the inner side of the thigh, or retraction of one or both testes. The blood is found uniformly diffused throughout the urine, so that the urine at the end of micturition has exactly the same characters as that voided first. Renal hæmorrhage usually imparts to the urine a characteristic brownish-red or dull smoky tint. The renal origin of the blood is, of course, unequivocally proved if there be tube-casts in the deposit. In most cases of renal hæmorrhage there is an entire absence of clots; when, however, the blood flows from the kidneys in considerable quantity, it coagulates, during its downward passage and is moulded into long vermicular clots in the ureters. The passage of these elongated worm-like casts gives rise to symptoms identical with those produced by a calculus passing along the ureter.

If the hæmorrhage come from the bladder, there is usually pain on pressure over the pubes, and marked disturbance of the vesical functions, with pain at the neck of the bladder before or after passing water, and undue frequency or difficulty of micturition. The urine comes away at first either of its natural colour or very slightly tinged with blood, while the latter portions frequently consist of pure blood, which, instead of being equally diffused through the urine, as in renal hæmaturia, is more or less clotted. These clots may impede micturition, and if retained in the bladder for any length of time may become colourless from the absorption of the blood pigment. When the bladder is distended with blood-clots, and retention of urine is thereby produced, it is almost certain that the bladder is the source of the hæmorrhage, as renal hæmorrhage seldom occurs to such an extent as to cause retention.

When the blood flows either *guttatim*, or in a stream apart from micturition, it most probably comes from some part of the urethra; it should be borne in mind, however, that if the source of the bleeding is near the neck of the bladder, the blood may regurgitate into that organ and so complicate the diagnosis.

Having thus indicated generally the means whereby the source of the blood may be determined, we are in a position to consider more in detail the various kinds of hæmaturia.

Hæmaturia from external injury.—The kidneys from their deep position in the body, are not very obnoxious to external violence, but sometimes they are injured by blows and falls on the loins and suffer lacerations, or they may be penetrated by punctured wounds. The bladder and urethra may also be ruptured by violence. In all these accidents blood may find its way into the urine. In the following case of laceration of the kidney from a fall, the writer had an opportunity of noting the condition of the urine from the time of the accident till the death of the patient five days afterwards. The patient was a bricklayer, aged 36, who, while intoxicated, had fallen from a height of seven stories, and was brought into the Manchester Royal Infirmary in a state of coma, with gasping respiration, having suffered a compound fracture of the skull. He passed no urine spontaneously after the accident, but a total of eleven ounces was withdrawn on three separate occasions by the catheter. The first specimen, obtained the day after the accident, was excessively bloody; it contained an immense number of dark granular casts evidently composed of crushed blood-clot; but there were no renal epithelial or pyelitic cells. The second specimen, obtained on the third day, was much less bloody, and contained, besides the dark granular casts, numerous deep brown casts with strongly-marked outlines and very few markings, and also a few transparent casts, some of which were studded with epithelium. In the third specimen, which was drawn just before death, and which was of a yellow colour and contained no fluid blood, a considerable deposit of chocolate-coloured blood granules took place; and although it was the product of forty hours' secretion, it only amounted to two ounces, with a sp. gr. of 1015. In the last two specimens free renal epithelium, browned by hæmatine, together with pelvic and infundibular epithelium, were present in abundance.

The autopsy revealed the existence of two lacerations on the posterior aspect of the right kidney, extending from the outer border almost to the hilum; they were completely closed by a firm wedge-shaped solid clot of blood, which was bleached where it came into contact with the substance of the kidney. The adjacent renal tissue seemed perfectly healthy. The lacerations did not extend to the infundibula, but two firm blood-concretions, one as large as a horse-bean, and the other the size of a pea, lay loose in the pelvis, with several smaller ones in the infundibula. No signs of inflammation were found in the brain or peritoneum. Death was evidently due, in this case, to suppression of urine.

Hæmaturia from violent muscular effort or straining.—Any great exertion, as in leaping, inordinate equestrian or pedestrian exercise, wrestling, and the like, may give rise to hæmaturia from engorgement of the renal vessels, and subsequent rupture of the vessels of the Malpighian tufts, or from rupture of congested vessels about the neck of the bladder. A case is cited by Dr. Beale in which hæmaturia occurred day after day in a healthy man just at the close of micturition, probably from the rupture of a few capillaries about the neck of the bladder consequent upon the effort to expel the last drops of urine. This disappeared after a time without any special treatment.

Hæmaturia from calculus in the kidney, ureter, or bladder.—This is a common cause of bloody urine. The position occupied by a calculus in the kidney influences to a considerable extent the amount of the bleeding. Thus so long as it remains where it was formed, there may be no symptoms leading to a suspicion of its presence, but immediately dislodgement has taken place, sudden pain sets in accompanied by more or less hæmorrhage. There is no direct proportion between the amount of hæmorrhage and the size and number of the calculi.

When minute calculous concretions are formed within the tubuli uriniferi, they sometimes give rise to hæmaturia coming on again and again, unaccompanied by any pain or discomfort save a slight aching or sense of fatigue in the lumbar region. On a careful examination of the urinary deposit, microscopic calculi of oxalate of lime or uric acid may be detected, with fibrinous casts dotted over with crystalline molecules, and there is more albumen in the urine than can be accounted for by the amount of blood passed.

Hæmaturia frequently takes place from the pelvis of the kidney or from the ureter, as a result of the passage of a calculus. When such is the case, there will be symptoms of pyelitis and nephritic colic. The blood, issuing from the pelvis, as a matter of course is not moulded in the renal tubes; it should be remembered, however, that clots may be formed in the infundibula or ureter, which may, in passing along the ureter, give rise to precisely the same symptoms as a calculus.

When vesical hæmaturia is caused by a calculus, the bleeding is usually inconsiderable; there are well-marked symptoms of vesical irritation, and the use of the sound will generally settle the diagnosis. When calculus is the supposed cause of hæmaturia the occurrence of a gravelly or crystalline sediment along with the blood would yield presumptive evidence in favour of this being the case. It is not to be considered conclusive, however, unless no other cause of hæmorrhage can be discovered. Dr. G. Johnson cites the case of a man who had hæmaturia as the undoubted result of the administration of half an ounce of turpentine for tape-worm, in whose urine blood-casts were detected studded with crystals of oxalate of lime.

In *hæmaturia from abscess, renal tubercle, embolism, and entozoa,* the quantity of blood is rarely abundant: the subject is treated of at greater length in the sections devoted to these affections respectively.

Hæmaturia from cancer of the kidney.—As a rule the hæmorrhage in renal cancer is irregularly intermittent and profuse, occasionally causing rapid anæmia and exhaustion; sometimes it is so trifling in amount that it cannot be detected without the aid of the microscope, and in more than half the cases it is altogether absent. In some cases it recurs at irregular intervals without any apparent cause, although its onset can occasionally be traced to some blow or fall. In two cases of hæmaturia from renal cancer under the observation of the writer, the presence of cancer-cells could not be discovered after repeated and careful examination. There is almost always a distinct renal tumour present in the anterior lumbar and hypochondriac regions which materially aids the diagnosis.

Renal congestion, active or passive may give rise to hæmaturia. Cases of this class will be considered more fully in the articles on congestion of the kidneys.

Hæmaturia from Bright's disease rarely takes place to an alarming extent. It is of most frequent occurrence in the earlier stages of the acute form, and the urine then contains, in addition to blood-corpuscles, abundance of tube-casts and renal epithelium. In the chronic forms still more than in the acute, copious hæmaturia is very rare. In these cases casts of various kinds will be found associated with the blood corpuscles, so as to leave little room for doubt as to the origin and cause of the hæmorrhage. The article on Bright's disease will contain further details on this head.

Morbid conditions of the bladder or prostate may give rise to the escape of large quantities of blood with the urine. The most common of these are cancer, villous disease, acute cystitis, and a varicose condition of the veins of the mucous membrane of the bladder. It should be borne in mind, too, that retention of urine in the bladder, whether arising from paralysis or from its not being fully evacuated by natural efforts, as in certain cases of stricture, may give rise to hæmaturia from decomposition of urea and generation of carbonate of ammonia which acts as an irritant.

Mental emotion has, in a very few recorded cases, been apparently the cause of hæmaturia. Thus Rayer mentions a case in which a fit of passion was followed by hæmaturia, and Dr. Basham[1] cites the case of a shoemaker who had frequently recurring attacks of hæmorrhage from the kidneys on each occasion of the misconduct of a drunken wife.

Supplementary Hæmaturia.—The cases included under this head are those remarkable instances where hæmaturia seems to take the place of some natural or morbid condition. Several cases are on record where a periodic discharge of blood with the urine seemed to be substituted for the menstrual flow. Chopart cites the curious case of a soldier, aged 19, who had a monthly discharge of bloody urine accompanied by all the symptoms characteristic of the menstrual flux. Rayer mentions two similar cases, one of which was that of a butcher of Sedan whose infirmity becoming known inspired so great disgust that no one would purchase meat from him. Chopart and Latour relate cases where a hæmorrhoidal flux was supplemented by hæmaturia; and the latter writer relates a case of spasmodic asthma which had proved so obstinate and severe that the patient had been unable to lie in bed for eighteen months, but which suddenly disappeared on the supervention of hæmaturia.

PROGNOSIS.—This depends upon (*a*) the amount and duration of the hæmorrhage; (*b*) the nature of the condition giving rise to it; and (*c*) the probable consequences of the hæmorrhage.

Hæmaturia is rarely fatal from the mere loss of blood, so that a favourable prognosis may generally be given *quoad* the hæmorrhage; indeed, in many cases, the occurrence of hæmaturia is a salutary process giving relief to the renal vessels when in a state of hyperæmia.

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[1] Basham on Dropsy, 3rd edit. p. 312.

The prognosis of hæmaturia is frequently merely the prognosis of the affection causing it, and will be favourable or unfavourable according to the nature of this affection.

The remoter *consequences* of hæmaturia are not unfrequently a source of danger. Blood effused into the uriniferous tubules coagulates and forms a series of plugs occupying their entire calibre; and unless these coagula are expelled by the pressure of the urine from behind, they continue to block up the tubes and thereby permanently destroy the function of the corresponding parts of the gland, and lay the foundation of a fatal renal degeneration.

Another occasionally untoward consequence of hæmaturia is the formation from the clot of a nucleus round which calculous concretions may form, so that hæmaturia may be the cause, as well as the consequence, of a calculous formation.

TREATMENT.—This obviously depends upon the cause, degree, and source of the hæmorrhage. When the cause and source are known, the treatment of the hæmaturia will be the treatment of its cause. Sometimes, however, cases occur in which we are unable to discover the cause, and the hæmorrhage is sometimes so excessive that direct attempts to check it must be made even although the cause be irremovable.

When hæmaturia is symptomatic of hyperæmia of the kidneys, such as that produced by acute Bright's disease, by overdoses of turpentine and cantharides, by external injuries or muscular efforts, it should not be interfered with unless it prove excessive, remembering that in such conditions a moderate bleeding is rather favourable than otherwise, as it relieves the congested vessels. Should it prove excessive, however, the best means of arresting it are rest in bed, and the diligent use of derivative measures, viz., the abstraction of blood by cupping over the loins, the promotion of free cutaneous transpiration by means of warm baths and diaphoretics, and the administration of hydragogue cathartics.

Passive hæmaturia occurring *in the course* of zymotic diseases should be carefully discriminated from acute Bright's disease occurring as a *sequela* of these affections. The former depends upon a general hæmorrhagic tendency, and the bleeding probably takes place from the whole urinary tract and not merely from the kidneys. The treatment ought to be constitutional, at least at first, and the remedies most efficacious in this class of cases are the mineral acids, especially sulphuric acid, freely administered. Where passive hæmaturia takes place from the bladder, copaiba and turpentine have been recommended; cautiously administered they are said to prove of service sometimes when other remedies fail. (Hassall.)

When hæmaturia is supplementary to hæmorrhoidal discharges, provided the blood be shed from the mucous membrane of the bladder and not from the substance of the kidney, it should not be looked upon as unfavourable, nor be rashly interfered with; should it become necessary, however, the application of a few leeches in the neighbourhood of the anus will give great relief. Where hæmaturia takes the place of the menstrual flow, it should be suppressed only on the re-establishment of the normal function.

In many cases of hæmaturia, however, we are called upon to treat it for itself, its cause being irremediable, as in cases of cancer of the kidney and bladder, villous disease, and the like. The two first indications in the treatment of such cases are the enforcement of perfect rest, and the application of cold to the bleeding part in the most effectual manner. Thus if the kidneys be the source of the blood, ice-poultices should be applied to the loins; if the bladder, iced water injections may be thrown into the bladder, and iced cloths applied to the perinæum and epigastrium.

Hæmostatics should also be employed; one of the most valuable of these is gallic acid, which may be given in five grain doses. frequently repeated. Dr. Golding Bird speaks highly of acetate of lead given frequently and in large doses for short periods. He recommends three or four grains, with one-fourth of a grain of opium in a pill every two hours, until six or eight doses have been administered, care being taken to keep the bowels open by saline purgatives. Ergot of rye has also been found of much service, and judging from the successful results of the hypodermic injection of ergotin in pulmonary hæmorrhages,[1] this method seems worthy of trial in hæmaturia. A solution of three to five grains of ergotin in ten minims of distilled water, or in five minims each of glycerine and rectified spirit, may be employed. Another valuable remedy is the tincture of the muriate of iron, which should be given in large doses frequently repeated. Dr. Johnson remarks: "This medicine arrests hæmaturia by a different process in different cases. Sometimes it has a directly astringent effect upon the bleeding vessels; but in other instances it acts by correcting the alkaline condition of the urine, which by its irritant action upon the bladder has excited the hæmorrhage." Alum, turpentine, and matico have also been found of service; turpentine should be cautiously administered on account of its irritating properties: its use is attended with less risk when the hæmorrhage is vesical than when it is of renal origin. Dr. Prout observes: "When the bladder becomes distended with blood, and complete retention of urine in consequence takes place, recourse must be had to a large-eyed catheter and an exhausting syringe, by the aid of which, and the occasional injection of cold water, the coagula may be broken up and removed. If the hæmorrhage be so profuse that the bladder becomes again distended with blood in a very short time, the injection of cold water into the rectum or bladder is sometimes of great use; and should these means fail, from twenty to forty grains of alum may be dissolved in each pint of water injected into the bladder, a remedy that seldom fails to check the bleeding even when the cause is malignant disease. I have never known any unpleasant consequences follow the use of this expedient; and have seen it immediately arrest the most formidable hæmorrhage when all other means had failed, and when the bladder had repeatedly become again distended with blood almost immediately after its removal."

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[1] Dr. Ritchie, *Practitioner*, vol. vii. 1871, p. 321.

ENDEMIC HÆMATURIA.

BY WILLIAM ROBERTS, M.D., F.R.S., F.R.C.P.

DEFINITION.—A peculiar form of hæmaturia indigenous to the Mauritius, Egypt, the Cape of Good Hope, Brazil, and other hot countries, associated with, and apparently due to, the presence of a minute parasite (Bilharzia hæmatobia), which infests the minute veins of the pelvis of the kidney, ureter, and bladder.

HISTORY.—This variety of hæmaturia seems to have been first recognised by Chapotin in 1812, as an affection endemic to the Isle of France. He has recorded several cases,[1] as have also Salesse,[2] Rayer[3] and Dr. Todd.[4] The real nature of the complaint remained unsuspected until the discovery of the offending entozoon, by Dr. Bilharz, while engaged conjointly with Griesinger, in investigating the diseases of Egypt.[5] It is however mainly by the researches of Dr. John Harley[6] on the endemic hæmaturia of the Cape of Good Hope, that the parasitic origin of that, and most probably of the endemic hæmaturia of all other hot countries, has been fully established.

PATHOLOGY AND SYMPTOMATOLOGY.—The entozoon discovered by Bilharz was named by him distoma hæmatobium; subsequent investigations, however, established the presence of a gynœcophoric canal which renders it generically distinct from the distomata. Diesing named it gynœcophorus hæmatobius; but the name by which it is most familiarly known is that given to it by Cobbold in honour of its discoverer, Bilharzia hæmatobia. It is a white, elongated, soft-skinned, bisexual entozoon, three or four lines in length, and belongs to the trematode order of worms.

The body of the male is comparatively thick and short, and is provided on its ventral aspect with a furrowed canal (canalis gynœcophorus) for the reception of the longer delicate filiform female during congress. The ovisac dehisces longitudinally; the eggs are about $\frac{1}{170}$th of an inch long, and are oval with an anterior terminal spiny projection. The newly-escaped embryo is flask-shaped and covered with cilia—by the aid of which it moves rapidly about.

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[1] Topographie Médicale de l'Ile de France. Paris, 1812.

[2] Sur l'hématurie, ou Pissement de Sang. Paris, 1834.

[3] Maladies des Reins, vol. iii.

[4] Clin. Lects. on Urinary Diseases, 1857, p. 62.

[5] Bilharz, Zeitschr. f. wissensch. Zoologie, Bd. iv. Griesinger, Beobachtungen über die Krankheiten v. Egypten. (Arch. d. phys. Heilkunde, 1854, p. 561.)

[6] Med. Chir. Trans. vols. xlvii., lii., and liv.

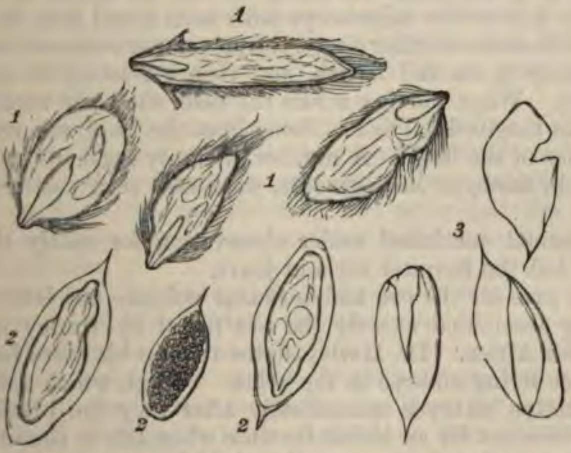
The first specimens were found by Bilharz in the portal vein and its branches and in the walls of the urinary bladder. The entire trunk of the portal vein is sometimes filled with it, the hepatic tissue containing numerous ova. The parasite does not however produce much derangement in the larger vessels. In the intestine, it is frequently associated with a disease resembling dysentery, giving rise to congestion and extravasation of blood, and frequently also to exudations on the free surface of the mucous membrane. The egg-shells have been found in the blood of the left ventricle. It is when lodged in the smaller vessels of the mucous and submucous tissue of the urinary tract, that the most pernicious effects of the parasite are seen. The bladder is chiefly affected; here it gives rise to injected and ecchymotic raised patches, varying from the size of a lentil to that of a shilling, covered with tough mucus or with greyish-yellow bloody exudation, containing masses of ova. At a later stage, the patches are more prominent and are mixed with minute specks of pigment; they may be smooth and leathery, or soft, friable, and covered with gravelly encrustations consisting of uric acid and other urinary deposits mixed with ova and blood. In some cases, the submucous tissue only seems involved, and the patches look like nodules or condylomata; in other instances, the mucous membrane may be thickened, injected, adherent, or detached. When the ravages of the entozoon extend to the ureter and pelvis of the kidney, still more dangerous consequences ensue. The thickening of the submucous tissue produces diminution of the calibre of the ureter. Above the constriction, dilatation of the ureter takes place in consequence of accumulation of urine, and a hydronephrotic condition is produced by the subsequent distension of the pelvis. In some cases inflammation and suppuration are set up and severe pyelitis supervenes, or renal disorganization and atrophy may ensue, as in a case observed by Griesinger in which the whole renal structure was destroyed, and the kidney had been converted into an enormous sac filled with pus.

Besides these direct results, masses of the parasitic ova are prone to become coated with urinary concretions, and thus to form the nuclei of large calculi; and it seems not improbable that septic infection may be produced by the accumulation of dead and dying animals in the portal vessels. Moreover they may creep into the general circulation and find their way into organs of vital importance. It is believed that pneumonia may thus be produced.

SYMPTOMS.—The first indications of the disease are the passage of a little dark-coloured blood after emptying the bladder of clear urine. The quantity of blood is increased after unusual exercise or travelling. At intervals, somewhat soft cylindrical filaments are passed usually stained with blood; sometimes they are nearly colourless. These red threads occasionally block up the urethra, and cause obstruction to the flow of urine. The quantity of urine voided is natural; occasionally a sudden sharp twinge of pain is experienced in the loins, which as suddenly subsides, sometimes a tickling sensation in the hinder part of the urethra is felt. Active exercise, such as riding, gives rise to pain in the back and perinæum. As a rule, the general health is good ; a feeling of lassitude is not uncommon, a cachectic or anæmic condition is sometimes met with.

The following case which fell under my notice in the Manchester Infirmary illustrates the main features of the disease as it is sometimes encountered in this country.

William Ray, aged 19, was admitted into the Manchester Infirmary under my colleague Dr. Simpson, in February, 1871. He stated that rather more than two years ago he went to Cairo, as groom, in the service of the Viceroy of Egypt. After a stay of some months, he went to Alexandria for the summer, returning to Cairo in the winter. He returned to this country about four months ago. While in Egypt he had been in the habit of drinking the water of the Nile unfiltered, and of eating water-cresses freely; with one or two exceptions, he invariably rode horses bare-backed. About four months after his arrival in Cairo, he observed that he passed bloody-looking urine, and shortly afterwards he suffered pain in the back and perinæum when riding. Since then he has persistently passed turbid urine containing blood. He is now very anæmic and thin, but in fair health apart from the urinary affection. On the 10th of March, I examined the patient's urine. The specimen was a fair sample of what he generally passed. It was smoky and turbid, with an abundant reddish-white deposit in which might be seen little flakes of blood-clots; it was



Bilharzia in the urine of Wm. Ray. 1. Free embryos—showing the different shape they assume as they swim about in the urine. 2. Ova containing unhatched embryos 3. Empty shells from which the ova have escaped.

neutral to test-paper, sp. gr. 1010, and contained a little albumen—rather more than the blood and pus would account for. Under the microscope, the deposit was seen to consist mainly of pus, mixed, however, with blood, both in the form of shreddy clots and as free corpuscles. Both free embryos and unhatched ova of the Bilharzia hæmatobia were present in considerable numbers (*see* Fig.) The embryos, in the mature ova, exhibited slow expanding and contracting as well as oscillating movements; these expansile movements were especially seen in the cervical narrowing, which at one time became very marked, and anon was almost effaced. The free embryos (1) moved actively in the urine for some hours after its emission. They were covered all over, with the exception of the head, with long vibrating cilia, by means of which they moved in various directions, with intervals of quiescence. At times an embryo could be seen racing across the field of the microscope with such speed that the eye could scarcely follow it—at other times, the movement consisted in stretching out the body to its full length, and then retracting it to an oval or ball-shape. When moving across the field, the body turned over and over on its longitudinal axis. Sometimes the head was retracted into the interior of the body. A number of empty eggshells, with irregular openings in them, or in fragments, were also seen scattered over the field (3).

This patient continued under observation for nearly three weeks, and then left the hospital without leave.

In this case all the ova had terminal spines—not lateral ones—so that they resembled exactly the ova found by Harley in the cases from South Africa. Dr. Harley states that in his cases he never met with a free living embryo in the urine. "Eggs, which split open and liberate active embryos immediately after they are placed in water, remain quiescent for an indefinite time when left in the urine, and all my attempts to hatch them in this fluid kept fresh and warm have invariably failed" (Med. Chir. Trans., vol. liv.). In the case just recorded this was certainly not the case—the embryos moved actively in the urine for several hours after its emission.

Etiological Considerations.—There can no longer be any reasonable doubt that the presence of this parasite in the urinary tract is the cause of these endemic forms of hæmaturia, but as to the precise manner in which it effects an entrance into the body there is still room for additional research. It seems probable that the most usual channel is the stomach, the ova or embryos of the parasite being swallowed in infected water or along with salad or vegetables. Dr. Harley suggests that it sometimes obtains admission through the skin—that the minute leech-like animal attaches itself to the skin of a person while bathing or wading, and implants its ova in some superficial vein. If such be the case, it is easy to understand that the hatching process and irritation attending the movements of the free embryos would result in an indolent form of ulceration, and that the little animals might be carried by the circulation from the limbs to the urinary organs. It is certain that new colonists in the Cape are prone to be affected, during the first year or two of their residence, with indolent sores on the legs, and it seems not unlikely that their origin may be thus accounted for.

There are a few other particulars relating to the etiology which call for remark. Endemic hæmaturia may occur at any age; but is most prevalent during childhood and young adult life. It seldom occurs under five or six years of age. Dr. Harley cites a case in a man of seventy-six. Different countries appear to be differently affected by it, in regard to the sexes. Thus Chapotin observes, "In the Isle of France, children of both sexes are affected from the tenderest age with hæmaturia;" while Dr. Rubidge of Port Elizabeth, writing to Dr. Harley, remarks, "Females are rarely affected, if at all. I have not myself observed a single well-marked case in this sex;" and Dr. Johnston says, "Hæmaturia prevails to some extent among the children of the civil community of Natal, and attacks both sexes, but boys more frequently than girls."

The disease seems incommunicable from husband to wife, as the wife of a patient of Dr. Harley had three or four healthy young children, and the husband had been passing numbers of the parasitic ova every day of their married life—yet the lady had never had the slightest symptom of the parasitic disease, and her urine was free from all traces of the parasite.

With regard to the distribution of the disease, it seems to be by far the most common in Egypt, where Griesinger found the parasite in 117 out of 363 autopsies performed by him. It is endemic in Natal, where it seems restricted to certain localities near the coast, the elevated regions of the interior remaining free from the parasite. In the Cape Colony, it appears to be limited to Port Elizabeth and Uitenhage.

PROPHYLAXIS.—This is a matter of great importance to the communities among whom the parasite is found, and it is suggested by our knowledge of the cause of the disease. From the researches of Siebold on the trematode worms, it may be assumed that between the ciliated embryo above described, and the adult sexual worm, there are two other distinct forms which serve to complete the chain of metamorphoses connecting these two extremes of development. Fresh-water mollusca and fish are probably the victims selected by the parasite during its development through these intermediate stages. Harley on these grounds suggests the following prophylactic measures in districts affected with endemic hæmaturia:—1. The water used in drinking should be conveyed from its source to its destination in covered channels, so that the ova contained in the excreta of those infested with the parasite may be prevented from mixing with it. 2. Drinking water should be filtered. 3. Salads which may entangle small mollusca containing parasites, and uncooked molluscs and fish (as smoked fish) should be carefully avoided.

PROGNOSIS.—This affection is rarely fatal in itself. In the case of children it seems to disappear gradually about the age of puberty, and apparently to entail no subsequent complications. Harley, however, remarks that although the hæmaturia ceases, the parasite probably remains and may give rise to an entirely new set of symptoms. Thus in one case under his notice hæmaturia had ceased for more than a year, and the patient believed himself perfectly free from any urinary or renal disorder, when he voided a small calculus. In another case, ten years after the disappearance of hæmaturia, calculi were passed at intervals, containing the ova of the parasite.

TREATMENT.—The remedies for ordinary hæmaturia have been tried in the endemic form without the slightest effect. They were given empirically with the view of checking the leading symptom. The rational treatment is to remove the cause, and in endeavouring to do this our efforts should aim at either killing or expelling the adult parasite, or, if this be impracticable, at securing the regular expulsion of the ova, which, so long as they remain inmates of the economy, are prone to become the nuclei of urinary calculi. Dr. Harley found that a draught composed of fifteen minims each of oil of turpentine and male fern, with five minims of chloroform in two ounces of tragacanth mixture given every morning, brought away great numbers of the ova; he relieved the renal irritation and pain due to the presence of crystalline concretions by giving the bicarbonate of potash in copious draughts of water. By this means the disintegration of calculi is facilitated and their formation prevented, as the uric acid, which he regards as the cementing medium of the deposits, is dissolved by the alkali.

When the bladder and prostate alone appear to be affected, we may have recourse to topical medication of the bladder. Dr. Harley tried the effects of injections of various substances into the bladder in the form of solution or emulsion. These were chiefly quassia, wormwood, oil of male fern, and iodide of potassium. The latter was the only substance which yielded satisfactory results. He recommends a solution of twenty to thirty grains of the iodide in five ounces of tepid water to be injected every second or third day, having found in a case reported by him that this "topical treatment was effectual in clearing away the accumulated products of the parasite, in securing their expulsion as fast as they were formed, and ultimately in destroying the adult parasite."

HÆMATINURIA AND PAROXYSMAL HÆMATINURIA.

By William Roberts, M.D., F.R.S., F.R.C.P.

Hæmatinuria.

Definition.—The passage of hæmatin, or the colouring matter of the blood, with the urine unaccompanied by rupture of the capillaries and the presence of blood-corpuscles.

Synonym.—False hæmaturia.

Pathology.—The urine is of a deep reddish brown or brownish black colour, looking very much as if it contained blood, but the most careful microscopical examination fails to reveal the presence of blood-corpuscles, or of fibrin. It always contains albumen, and coagulates on boiling. Vogel gives the following explanation of the appearance of free hæmatin in the urine. By the decomposition of blood-corpuscles which is constantly taking place in the body during the normal nutritive changes, hæmatin is set free, and after subsequent metamorphoses, is probably excreted in the form of urinary and biliary pigment, so that in health hæmatin never directly passes into the urine. But in certain diseased conditions, excessive disintegration of the red blood discs takes place, and the quantity of hæmatin thereby set free is too large to undergo the normal changes, and a portion of it is therefore excreted unchanged in the urine. This rapid destruction of red-corpuscles occurs in affections associated with a so-called "dissolved state of the blood," in septic, pyæmic, and putrid fevers, in some extreme cases of scurvy and purpura, and a temporary, but most marked, hæmatinuric condition is produced by the inhalation of arseniuretted hydrogen. Vogel relates a case in which the urine was of an inky blackness for about twenty-four hours after the inhalation of arseniuretted hydrogen during a chemical experiment. He has further shown by experiment that the same condition may be produced artificially in animals by causing them to inhale arseniuretted hydrogen and also carbonic acid, and by the injection into the veins of substances known to produce disintegration of the blood-corpuscles.

The existence of hæmatinuria indicates an excessive decomposition of blood-corpuscles; it admits of a favourable prognosis if the cause be of a transient nature; should it appear in the course of scorbutic or septicæmic conditions, however, it must be looked upon as a sign of great danger to the patient.

PAROXYSMAL HÆMATINURIA.

DEFINITION.—A disorder occurring in paroxysms characterized by the sudden onset of a feeling of cold or shivering, genererally after some exposure, accompanied by a dull sense of weight or pain in the lumbar region, and terminating, after an interval varying from half an hour to two hours, with the discharge of very dark bloody-looking urine, in which, however, no blood corpuscles can be detected. The urine at the next micturition or the one after is found to present its normal appearance. These paroxysms recur at irregular intervals, and are never followed by a hot or sweating stage.

SYNONYMS.—Intermittent Hæmaturia (Harley); Winter Hæmaturia (Hassall); Intermittent Hæmatinuria (Gull); Paroxysmal Hæmaturia (Pavy); Intermittent Cruenturesis (Thudichum).

HISTORY.—This curious affection seems to have escaped recognition up till a very recent period. Rayer, in his comprehensive treatise on diseases of the kidneys, cites three cases which resemble in some particulars the affection under consideration; none of these cases came under his own observation, however, and it is doubtful, in the absence of chemical and microscopic examination of the urine, whether they really were cases of paroxysmal hæmatinuria.

We are indebted to Dr. George Harley[1] for the first accurate description of this disorder.[2] He published two cases which had come under his observation; he was followed very shortly afterwards by Dr. Dickinson,[3] who related the particulars of four cases. Cases have been subsequently published by Gull,[4] Murchison,[5] Pavy,[6] Greenhow,[7] Hassall,[8] Habershon,[9] and others.

The present writer has collated these cases, to which he has added three others, two of which were under his own observation and one under that of Dr. Ritchie; from an analysis of these, twenty cases in all, the following account is drawn up.

[1] Med. Chir. Trans. vol. xlviii. p. 161.

[2] Dr. Wickham Legg has directed attention to the fact that Dessler, in 1854, published a full account of this disorder in Virchow's Archiv for that year. See Dr. Legg's paper, St. Barthol. Hosp. Rep., vol. x.

[3] Ibid. p. 175.

[4] Guy's Hosp. Reports, 3rd Series, vol. xii. p. 381.

[5] Path. Soc. Trans. vol. xvi. p. 183.

[6] Ibid. vol. xviii. p. 157.

[7] Clin. Soc. Trans. vol. i. p. 40; and Edin. Med. Journ. vol. xiii. p. 996.

[8] Lancet, 1865, vol. ii. p. 369.

[9] Ibid. 1870, vol. i. p. 158.

CLINICAL HISTORY.—The symptoms of a typical case of paroxysmal hæmatinuria are as follows:—An individual is exposed to cold, or to damp and cold conjoined; he suddenly experiences a feeling of coldness of the extremities, and this is followed by general chilliness, passing into distinct rigors, accompanied by a feeling of malaise, a disposition to stretch himself and to yawn. An uneasy sense of weight, or a dull heavy pain is felt in the loins, extending round to the umbilicus and passing down the thighs; there is pain or a feeling of weakness or stiffness in the lower extremities, nausea, and, perhaps, vomiting. There may be retraction of the testicles. About an hour or a little more from the commencement of the paroxysm, a quantity of dark porter-like urine is passed, after which the pain and general disturbance subside, leaving the patient apparently perfectly well until the next paroxysm. Each paroxysm lasts from three to twelve hours, and it is worthy of notice that no paroxysms occur at night, the urine voided before breakfast being invariably natural. The recurrence of the paroxysms in different cases, is most variable. In some instances, they recur once, twice, or even thrice a day; more commonly they recur on alternate days, or twice a week, or once in ten days, or quite irregularly. They may recur in this way with more or less regularity during a period of a few days, or five or six weeks, and then cease altogether for a few days, or for weeks or months, and recur again in a similar manner. The disorder may thus continue an interrupted course for many months or years; in one case cited by Dr. Dickinson, the paroxysms had recurred at varying intervals during eleven years. After the disease is established the subsequent paroxysms are usually (but not always) quite unconnected with a fresh exposure to cold or damp.

Characters of the Urine.—The colour of the urine resembles that of porter or of the darkest port wine; it is generally turbid, and deposits, on standing, an abundant chocolate-coloured sediment. The specific gravity varies from 1015 to 1033, usually ranging between 1022 and 1025; the reaction is usually acid, but it may be faintly alkaline. It is always highly albuminous, and on boiling, the albumen coagulates into brownish masses, which on subsiding leave the clear supernatant urine of nearly its original dark red colour. Increased elimination of urea takes place during the paroxysms: in one of Dr. Harley's cases, the amount of urea in a specimen of urine passed during the paroxysm was 3·6 per cent., and the next urine passed after the paroxysm only contained 1·8 per cent. In a case observed by Dr. Dickinson, the urea reached 4·8 per cent., whilst in a specimen examined by the writer, it amounted to so much as 5·2 per cent.

The chocolate-coloured sediment, when examined microscopically, is found to consist chiefly of amorphous granular matter, which is presumably the product of disintegrated blood corpuscles. There are also present a few casts of tubes, some of which are transparent and fibrinous, but they are mostly of dark granular appearance. Crystals of oxalate of lime are generally seen, and occasionally amorphous urates are present: *very rarely indeed* a few stray blood-discs. Sir William Gull found myriads of minute crystals of hæmatine having a brilliant red colour.

The change in the urine may take place with the utmost abruptness, that passed at one micturition being porter-like, and possessing the characters just enumerated, and at the next, straw-coloured, free from albumen, and in every respect normal; or it may change more gradually, resuming its normal appearance at the fourth or fifth micturition after the paroxysm.

THE STATE OF THE GENERAL HEALTH.—In most cases the patient is sallow and pale;[1] sometimes hepatic derangement has been present when the patient came under observation. Of the twenty cases collected, four had at some time suffered from ague; but in the remainder there was no evidence or suspicion of ague or exposure to paludal influences. Many of the cases seem to have the rheumatic diathesis, and in one case under the observation of the writer, subacute rheumatism set in with swelling and pain in the joints after the cessation of the paroxysms. Bronchitis and asthma have been concomitants in one or two cases.

PROGNOSIS.—This is generally good. Of the twenty collated cases none died; twelve were reported as having completely recovered, one was convalescent, and seven were in progress at the time they were reported. By complete recovery is understood that the paroxysms did not recur for a period varying from six weeks to four years; it ought, however, to be borne in mind that a relapse may take place after several months—in one case the paroxysms recurred in five months, and in another, two years after, they had entirely ceased.

ETIOLOGICAL AND PATHOLOGICAL CONSIDERATIONS.—The liability to this affection seems to be almost exclusively confined to males, as out of the twenty collected cases only one occurred in a female. The *age* of the patient at the time of the invasion ranged between two years and forty-eight; two cases occurring under twenty, seven between twenty and thirty, six between thirty and forty, two between forty and fifty; in three instances the date of invasion is not specified.

The *exciting cause* was distinctly connected with malarial poisoning in two cases; in all the others, with one exception, the disorder was clearly attributable to vicissitudes of temperature or exposure to damp. The effects of exposure to cold are well exemplified in a case of Dr. Johnson's, in which the patient, so long as he remained in bed, continued free from the paroxysms; but if he sat up and got chilled a paroxysm came on; in a case mentioned by Dr. Pavy, the patient sometimes averted an attack by going indoors directly he felt it coming on, and sitting before the fire and drinking something warm. Sir William Gull believes a blow or injury to the loins may produce this disorder, and cites the case of a young lady who fell on her back in getting into a railway carriage, and passed, shortly afterwards, dark, bloody-looking urine, which contained "only the granular pigment matter of disintegrated blood-corpuscles." In the absence of farther evidence, however, we must hesitate to accept such injuries as a cause of *paroxysmal* hæmatinuria.

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[1] In one case, reported by Dr. Dickinson, the patient had the appearance of robust health the day before an attack came on.

The *pathology* of the affection is at present very obscure; it seems clear, however, that the kidneys are affected, the symptoms indicating a sudden transitory congestion of the renal capillaries with escape of their contents, *without rupture of their walls.* We may infer that this is produced through the medium of the nervous system, from the paroxysmal nature of the disorder and the nervous phenomena which characterize its onset, *viz.*, the rigors, nausea, and malaise. There seems to be some hitherto unknown connection between this disorder and ague; but though related, they are not identical, for in by far the greater number of the recorded cases, there was no aguish tendency nor evidence of exposure to malaria. Besides, there are important differences in the course of the two affections: thus, so far as the temperature has been observed in paroxysmal hæmatinuria during the initial stage, it has been, unlike what obtains in the cold stage of ague, considerably lower than normal; moreover, no distinct hot or sweating stages, and no constant periodicity are observed in the hæmatinuric paroxysms.

TREATMENT.—The remedies appropriate to hæmaturia are perfectly inefficacious in this affection. In two of the recorded cases the attacks seem to have passed off without medicinal treatment, simply by avoiding exposure to cold; in a case observed by the writer, the paroxysms gradually subsided under the use of a pill containing one grain each of quinine and sulphate of iron. In one of Dr. Dickinson's cases, cupping over the loins, vapour baths, gallic acid, quinine, and iron in various forms, were tried in succession, but nothing seemed to affect the disorder: "the hæmorrhage always ceased on the removal of the cold which caused it." Dr. Harley gave mercurials, and afterwards quinine, in his cases, with marked benefit, one patient having remained free from the paroxysms for four years' during which he was under observation. Sir W. W. Gull gave two-drachm doses of compound tincture of cinchona three times a day with benefit; and Dr. Hassall found that the hæmorrhage was considerably restrained by giving, night and morning, a powder containing tannic and gallic acids and burnt alum, with a mixture containing quinine, sulphate of iron, and excess of sulphuric acid during the day. Dr. Habershon found that quinine and arsenic, and Dr. Greenhow that quinine and perchloride of iron, and afterwards quinine and syrup of the iodide of iron with iodide of potassium, had the effect of causing the urinary symptoms to subside, and the patients remained free for several months afterwards during the time they were under observation. Dr. Beale[1] calls attention to the importance of giving quinine in full doses, not less than six grains, in order to combat the disorder successfully. During the paroxysm, Dr. Ritchie found the best treatment was to send the patient to bed, apply artificial heat, and administer warm stimulating drinks, such as hot brandy-and-water.

The evidence generally is strongly in favour of quinine and iron as the most effective medicinal agents.

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[1] Practitioner, vol. i. 1868, p. 73.

ALBUMINURIA.

By William Roberts, M.D., F.R.S., F.R.C.P.

Normal urine may be said to be entirely free from albumen. In certain cases, however, albumen has been observed temporarily in the urine of apparently healthy persons. This may be due to dietetic errors, as in the case of Barreswil, who passed albuminous urine for twenty-four hours after eating ten eggs;[1] or it may be due to dyspepsia, as in the case of Beneke,[2] who four times during so many weeks detected albumen in his own urine while suffering from dyspepsia. Eleven years after, he remained perfectly free from indications of renal disease.

The presence of albumen in the urine is not so invariably associated with kidney disease as was once supposed; but is caused by a variety of conditions, a correct appreciation of which is essential to a proper understanding of its pathological import. As albumen is the most common and the most important of the abnormal ingredients found in urine, it is obviously necessary that its presence should be looked for in every specimen of pathological urine.

Tests for Albumen.—The practical tests for albumen are coagulation by heat or by nitric acid; in doubtful cases it is necessary to combine these two tests.

Heat.—When urine contains albumen, and possesses its usual acid reaction, it begins to get turbid when heated to 140° F., and on the temperature being raised to boiling the albumen coagulates in flaky masses, which become more compact the longer the heat is applied. If the urine contain a large quantity of albumen it will coagulate at a somewhat lower temperature than a feebly albuminous urine, and if only a trace be present, no turbidity will take place until the urine begins to boil. This turbidity persists after the addition of an acid. When the urine is cloudy from the precipitation of amorphous urates (a certain indication of acid reaction), heat *alone* is a complete test for albumen; it is moreover the surest test. The best way of proceeding to test for albumen by heat is to fill a test-tube to the depth of about an inch with the suspected urine and to apply the flame of a spirit-lamp to the upper portion of fluid. If the urine be clear, the lower portions preserve their original transparency, while the upper portions (if albumen be present) become turbid; if the urine be cloudy from urates three distinct zones become visible when albumen is present: the upper one is turbid from the coagulated albumen; the middle is clear from the dissolution of the urates, which disappear at a lower temperature than is necessary to coagulate albumen; and the lowest portion, being unaffected by the heat, remains cloudy and unchanged. There are two points which it is necessary to remember when heat alone is employed as a test for albumen. The first is that when the urine is alkaline, albumen is not coagulated by heat; so that the acidity must be restored before the application of heat: this may be readily effected by the addition of a few drops of acetic acid. The second point is that a neutral or alkaline urine becomes turbid on heating from the precipitation of earthy phosphates; but this turbidity may be readily distinguished from that due to coagulated albumen, as the phosphates immediately disappear on the addition of a drop of any acid.

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[1] L'Union Méd., 1857, No. 155.

[2] Archiv des Vereins für wiss. Heilk. Bd. I.

Nitric Acid.—This is the most delicate test for detecting minute quantities of albumen. The best way of using it is as follows: Fill a test-tube with the urine to the depth of about an inch, then, inclining the tube, pour in very slowly strong nitric acid in such a manner that it may trickle along the sides of the tube to the bottom, forming a stratum about a quarter of an inch deep below the urine. If it is added in this manner, there is scarcely any commingling of the two fluids. If the urine be albuminous, three layers will be observed in the test-tube: at the bottom there is the colourless nitric acid; immediately above, a zone of opalescence from the coagulated albumen; and the unaltered urine above this. When only a trace of albumen is present the opalescent zone is not visible for several minutes. If the acid is not added in the manner just described, the *nitric acid test* is liable to two fallacies: for if the urine be feebly acidulated by the addition of only a drop or two of nitric acid, coagulation of the albumen is thereby prevented; while, on the other hand, if a large quantity of acid, say an equal volume, be suddenly added to the urine and mixed with it, no turbidity takes place, although the urine be highly albuminous. The reaction of the urine does not interfere with this method, but it is important to remember that in concentrated urines, and especially in febrile urines, the addition of the acid is liable to precipitate the amorphous urates, thus producing a turbidity which might be mistaken for albumen. To discriminate between these conditions, it is necessary to observe the level at which the cloudiness begins, and the direction in which it extends. Thus the turbidity due to coagulated albumen begins immediately above the stratum of acid and extends upwards; while that due to urates appears first at or near the surface of the urine, and spreads downwards. The application of heat also serves to distinguish the two reactions; for the urates are speedily dissolved when heat is applied, while the turbidity due to albumen remains unaffected by heat.

The slight opalescence which is commonly present in the urine of persons taking copaiba and cubebs is sometimes, but not always, increased by nitric acid in the cold; but the odour of these drugs is readily detected, while the application of heat diminishes the opalescence and prevents any turbidity with nitric acid. Nitric acid in the cold causes in urines rich in urea a slow precipitation of crystals of nitrate urea, which however is so unlike coagulated albumen that it can scarcely be mistaken for it.

The following practical directions may then be followed in proceeding to test for albumen: when the urine is cloudy from urates, use heat alone; when the urine is clear, or in any case if it be alkaline, add nitric acid in the way just described; if no turbid zone appear above the layer of acid, the urine may be certainly pronounced free from albumen; if, on the other hand, a turbid zone is produced, which is not dissipated on the application of heat, albumen is certainly present.

A number of other substances possess the property of coagulating albumen, among which may be enumerated alcohol, bichloride of mercury, sulphate of copper, alum, ferro-cyanide of potassium, and creasote; but they are unsuitable for detecting albumen in the urine, because they precipitate at the same time some of the natural constituents of the urine.

The *quantitative* estimation of albumen in urine is a matter of considerable practical importance, and various plans of obtaining this object have been devised.

For precise determinations the plan usually followed is to bring a measured quantity of urine to a slightly acid condition; boil; throw on a weighed filter; wash; dry at 212°; and weigh. This proceeding demands a good deal of time. The filtering is sometimes impossible; and the results obtained are only moderately accurate with every care.

For a rough and ready, but useful, method, there is none superior to boiling the urine in a test-tube and then adding a drop or two of acetic acid. The albumen coagulates in flakes, and presently sinks to the bottom, forming a layer of various thickness. The proportion of albumen is judged of by the depth of this layer as compared to the height of the column of urine in the tube. This proportion may be expressed in numbers, as $\frac{1}{2}$, $\frac{1}{4}$, $\frac{1}{12}$ and so forth. If the quantity of albumen be too small to form a layer of appreciable depth, the proportion is expressed more loosely, as a "cloudiness" or an "opalescence." The varying density of albuminous urines, and the varying size of the flakes into which albumen coagulates, affect the rapidity and completeness of the subsidence, and therefore the depth of the coagulated layer, so that only approximate results can be expected from this method.

Becquerel ingeniously turned to account the property of albumen to deviate the plane of polarisation to the left, and constructed an instrument on a similar plan to the optical saccharimeter, by which the deviation could be measured and the percentage of albumen calculated therefrom. It would appear, however, that this instrument, on Becquerel's own showing, is only capable of very limited clinical application. When the quantity of albumen is considerable it gives very exact indications; but the deviation is too slight for exact estimation in moderately and feebly albuminous urines; it is therefore useless for the bulk of albuminous urines.[1]

Boedecker has recently proposed a volumetrical method, founded on the property of ferrocyanide of potassium to form an insoluble compound of fixed composition with albumen. Vogel states that he has found this method inaccurate.[2]

New Process.—Dilution Method.—The writer has recently proposed a mode of estimating albumen in urine, which he thinks will prove very useful in clinical work.[3] The principle of the method is easily understood.

When an albuminous urine is progressively diluted with water, and tested from time to time with nitric acid, the opacity induced by the test becomes gradually fainter and fainter, until at length it ceases to be visible. This point is reached when the urine contains less than about 0·0014 per cent. of albumen. The more albumen the urine contains, the more dilution, of course, it will require to reach the vanishing point of the reaction; and if we could fix this point with accuracy we should have a simple method of estimating albumen in urine. The urine could be diluted until it ceased to react with nitric acid, and the amount of dilution required to reach this point would furnish a measure of the proportion of albumen.

But it is not possible to fix this point with accuracy. The opacity produced by the acid fades away so gradually with increasing additions of water, that it is impracticable to decide within many degrees the point at which the reaction ceases to be appreciable. And not only so, but the development of the reaction becomes more and more retarded as the dilution proceeds, until at length it only becomes visible after the lapse of several minutes.

To overcome this difficulty it was found necessary to fix on some arbitrary point or line which would serve as a practicable zero to the scale. After many trials it was found most convenient to draw the line at a reaction coming into sight midway between half and three-quarters of a minute after the contact of the acid—that is, to dilute the urine until it gives no reaction for thirty seconds after the addition of the acid, but shows a distinct opalescence at the forty-fifth second. The exact point to be aimed at is a reaction coming doubtfully into view between the thirty-fifth and fortieth second, and appearing, still very dim, but unmistakable, at the forty-fifth second. It was found possible, after a little practice, to strike this point with sufficient exactness to serve as a practical zero to the scale.

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[1] See a clinical lecture by Becquerel, Clinique Européenne, 1859.

[2] Boedecker's method is described in Henle and Pfeufer's Zeitsch. 1859, p. 321.

[3] For a fuller account of this method the reader is referred to a paper by the author, read before the Medico-Chirurgical Society, Feb. 22, 1876.

Each dilution with an equal volume is counted as one degree on the scale, and these degrees may be conveniently termed "degrees of albumen." Thus a urine requiring dilution with forty times its bulk of water to reach the zero-reaction may be described as possessing forty degrees of albumen—a urine requiring three hundred similar dilutions as possessing 300 hundred degrees of albumen, and so forth.

The difficulty of the method is to hit correctly the zero-reaction. When this point is approached, a little more or a little less dilution makes but a slight difference in the time at which the reaction appears. In order, therefore, to obtain exact results, it is necessary to conduct the testing with rigid uniformity. The test-tube employed should have an interior diameter of $\frac{5}{8}$ of an inch (15 millimetres); the acid must be added in the right way, and at the right moment. The operation, too, should be performed by daylight, or, if by gaslight, an addition of about five per cent. must be made to the results. The proceeding adopted is as follows:—The urine is first tested in the usual way with nitric acid, so as to get a rough idea of the quantity of albumen contained in it, and of the degree of dilution likely to be required to reach the zero. The watch is placed on the table before the operator. A fluid drachm of the urine is then measured off and introduced into a graduated pint measure, and water is added up to a few or many ounces, according to the degree of dilution likely to be required to approach the zero-reaction. The test-tube is then filled to the depth of about an inch with the diluted urine, and held widely inclined from the perpendicular. The eye is now directed to the watch, and the acid is added in such a manner that it runs along the lower side of the tube to the bottom and forms a distinct layer, about a quarter of an inch deep, below the diluted urine. The acid must be added exactly on one of the quarter-minute strokes. This is the most critical step in the proceeding, and it should be performed in the following manner:—A pointed glass tube or pipette is dipped to the depth of a couple of inches into the acid and covered with the forefinger. The pipette, thus guarded, is then passed into the test-tube to within half an inch of the level of the diluted urine, and at the right moment the finger is removed and the charge of acid delivered. As soon as the acid is added the test-tube is held up to the light against some dark background (such as a black sleeve, a book bound in black cloth, or a dark corner of the room), and as soon as the faintest opalescence is perceived above the level of the acid, the time of its appearing is noted. If this appear at or before thirty seconds after the contact of the acid, more water is added, and the testing repeated as before. Thus, by successive additions of water and repeated testings, a close approximation to the zero-reaction is obtained. A fresh dilution is then prepared, and, guided by the previous trials, two or three testings with different dilutions are generally sufficient to indicate with exactness the dilution which produces an opalescence between the thirty-fifth and forty-fifth second after the addition of the acid.

If too much water is added in the first instance the reaction does not appear until after the forty-fifth second. In this case the operation must be re-commenced with less water, and proceeded with as in the first case.

When the zero-reaction is determined, the degree of dilution required to produce it is noted, and expressed in multiples of the unit-volume of urine employed. Thus, if a fluid drachm was the unit-volume of urine employed, and the zero-reaction was obtained when dilution was carried up to fifteen ounces (120 drachms), the urine is recorded as having 120 degrees of albumen.

If the urine is feebly albuminous—indicating less than 20 degrees of albumen—the fluid ounce should be substituted for the fluid drachm as the unit-volume. On the other hand, if the urine indicate more than 160 degrees of albumen, the unit-volume should be half a drachm—or, still better, the urine should be previously diluted with water in the proportion of 1 in 2 or 1 in 4, and the result afterwards multiplied by 2 or 4 as the case may be.

The actual value in weight of albumen of each degree on the dilution scale was found, by careful comparative experiments with the weighing method, to correspond to 0·0034 per cent., or 0·0148 grain per fluid ounce of the British Pharmacopœia. These data supply an easy means of calculating the quantity of albumen per ounce, and also the daily loss of albumen. Suppose that 40 ounces of urine were voided in twenty-four hours, and that a sample of this urine showed 150 degrees of albumen by the dilution method, then —

$$0{\cdot}0148 \times 150 = 2{\cdot}22 \text{ and } 2{\cdot}22 \times 40 = 88{\cdot}8.$$

The urine contained 2·22 grains of albumen per ounce, and the daily loss was 88·8 grains.

The time required for the estimation of albumen in urine by this method is from ten to twenty minutes.

Modified Albumen in Urine (*Paralbumen*).—Dr. Bence Jones has described a modification of albumen found by him in the urine of a patient suffering from mollities ossium. He regarded this substance as the hydrated deutoxide of albumen. The urine in which it was found was slightly acid, and of sp. gr. 1034·2; another specimen had a sp. gr. of 1039·6; it did not give a precipitate with nitric acid alone, nor by boiling, nor by adding nitric acid to the boiling urine. If however the urine was boiled and then allowed to cool, a precipitate was formed, which was immediately redissolved by heat.

The same substance has been found in the buffy coat of the blood in inflammation, and it is also met with in the albuminous fluid of pus.

Dr. Moore has published a case[1] in which albumen assumed a distinctly caseous modification in the urine for a short period. The urine when first examined contained albumen in its usual state; but that voided the following day coagulated with acetic acid, and formed repeated pellicles when evaporated. Seven days afterwards the urine contained no casein. In a case observed by Dr. Leared,[1] a modification of albumen, different from either of the above, was encountered. The urine became turbid with nitric acid, and again became clear when sufficiently heated. It behaved in the same way when heated with hydrochloric and phosphoric acids.

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[1] Med. Times and Gaz., 1866, ii., 525.

Clinical Significance of Albumen in the Urine.—In discussing this point it is necessary to exclude all those cases in which albumen is merely incidental to the presence of some other fluid in the urine, such as blood, pus, or, more rarely, semen. The excessive use of a too exclusively albuminous dietary, such as eggs, has been shown by Barreswil, Brown-Séquard, and others, to cause the urine to become slightly albuminous. Claude Bernard found that irritation of the renal nerves and of a certain spot in the floor of the fourth ventricle, higher up than the diabetic puncture, caused albumen to appear in the urine of animals. The same result followed the injection of the albumen of eggs, and even of a large quantity of pure water into the veins (Magendie). Any interference with the renal circulation produces albuminuria, as will be shown in the section devoted to Congestion of the Kidneys. Vogel found that inhalation of arseniuretted hydrogen and carbonic acid caused the urine to be abundantly albuminous. The notion that mercurial salivation produced albuminuria was long ago shown to be erroneous by Rayer; and his observations have been since confirmed by those of Dr. Francis, who examined the urine of fifteen salivated persons without finding a trace of albumen.[2] Slight and temporary albuminuria seems to occur in very exceptional cases apart from dietetic errors, from very slight disorders, as in the case of Beneke previously referred to. Similar observations have been made by Clemens, Rayer, and Solon. With these unimportant exceptions, albuminuria must always be looked upon as a grave symptom of disease; and when its presence is established, it becomes of the utmost importance to the practitioner to know its true significance.

The pathological states in which albumen appears occasionally or constantly in the urine may be grouped under the following heads:—

1. Acute and chronic Bright's disease of the kidneys.
2. Pregnancy and the puerperal state.
3. Febrile and inflammatory diseases.
4. Impediments to the circulation of the blood.
5. A hydræmic and dissolved state of the blood and atony of the tissues; also hæmatinuria.
6. Saturnine intoxication.

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[1] Ibid., 1871, ii.

[2] "Diseases of the Kidneys," by G. O. Rees. Lond. 1850, p. 28.

In the *first group* albuminuria is always, and in the *second* frequently, dependent upon structural changes in the kidneys, and the reader is referred to the article on Bright's disease for the consideration of these heads in detail.

The *third group* comprises zymotic diseases such as scarlet fever, measles, small-pox, typhoid fever, cholera, yellow-fever, ague, diphtheria, &c.; and inflammatory diseases such as pneumonia, peritonitis, traumatic fever, articular rheumatism, &c. In all such complaints a trace of albumen is occasionally found in the urine; it usually amounts to no more than a trace, and disappears on defervescence; sometimes the quantity is large in pneumonia.

In zymotic diseases there is a twofold pathological condition, viz. pyrexia and the operation of a specific poison; and albumen may appear in the urine either incidentally to the febrile state, when it is comparatively unimportant, or as an indication of serious structural changes in the kidneys, constituting a grave sequela of the disease. As intercurrent febrile attacks are common in the course of most chronic complaints, temporary albuminuria has been observed in very many different diseases—especially in chronic tuberculosis, cancer, caries, and necrosis; under such circumstances the albuminuria must be carefully discriminated from that produced by co-existing genuine Bright's disease.

The *fourth group* includes impediments to the circulation of blood from emphysema, heart disease, abdominal tumours, cirrhosis, &c., and will be discussed in the article on Congestion of the Kidney.

In the *fifth group*, albumen appears in the urine unconnected with organic changes in the kidney. It occurs in purpura, scurvy, pyæmia, hospital gangrene; it is also associated with the escape of the colouring matter of the blood and often with jaundice. (*See* Hæmatinuria.)

In the *sixth group*, albuminuria is due to saturnine intoxication. Albumen had long since been occasionally found in the urine of persons suffering from lead-poisoning, but it had been looked upon as a mere coincidence, until Ollivier[1] by experiment and clinical observation demonstrated their connection as cause and effect. Thus he found that the urine of dogs, rabbits, and guinea-pigs poisoned with repeated doses of carbonate of lead became albuminous, and that their kidneys showed signs of incipient organic disease. He also collected fifteen cases of albuminuria in persons poisoned with lead, seven of whom had temporary albuminuria; in three, the albuminuria persisted during the continuance of the plumbism; and in four, genuine Bright's disease had been produced. He also examined the urine of 37 persons with various manifestations of lead-poisoning in l'Hôpital de la Charité: of these, nine had albuminous urine. He found that both the urine and the kidneys in these cases contained traces of lead; and he inferred that the presence of lead in the kidneys induced an organic lesion of these organs, and that the albuminuria was the consequence of that lesion. These observations have been confirmed by Lanceraux[1] and Danjoy.[2]

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[1] Archives générales, 1863, ii., pp. 530 and 709.

When albumen is ascertained to be present in the urine, the important question to determine is, whether or not it indicates the existence of organic disease of the kidneys; and this can only be decided after a careful consideration of the three following points, namely:—

1. The temporary or persistent duration of the albuminuria.
2. The quantity of the albumen; and the occurrence and character of a deposit of renal derivatives.
3. The presence or absence of any disease outside the kidneys which will account for the albuminuria.

1. With the view of drawing a stronger line of demarcation between temporary and permanent albuminuria, Dr. Parkes[3] analysed the adult cases treated by him in University College Hospital, in which the urine was examined carefully and daily for a sufficient length of time to enable it to be said with perfect certainty that albumen was or was not present during the whole course of the disease, or during any part of it. Cases of cystitis and vaginitis were not included: the cases were of the miscellaneous character usually admitted into a London general hospital. All cases of cholera and of pregnancy were excluded from consideration. Under the heading "Temporary Albuminuria," Dr. Parkes included cases in which albuminuria, after lasting some days, or even weeks, disappeared entirely for some time before the patient left the hospital; and under "Permanent Albuminuria," cases in which the albumen did not disappear during the time the patient was under observation—this time being generally very long, and always many days. The total number of cases tabulated was 303—170 men and 133 women. He found the proportion of cases of temporary albuminuria almost the same in the two sexes, viz. rather over 12 per cent.; while the proportion of cases of permanent albuminuria in males was 14·7 per cent. as against 10·5 in females: this difference is attributed to the greater prevalence of organic kidney disease in male hospital patients in London. The entire percentage of albuminous cases, both temporary and permanent, was in men a fraction over 27 per cent., and in women $22\frac{1}{2}$ per cent. This percentage is considerably greater than that given by Dr. Barlow, who found that only 9 per cent., out of 300 cases investigated by him, had albuminuria. Of the 37 "temporary" cases, the largest number occurred in acute Bright's disease, pneumonia (acute, lobar), acute rheumatism, and typhoid fever. The quantity of albumen was "large" in the cases of pneumonia and acute Bright's disease; was in "some quantity" in the cases of typhoid, variola, and scarlatina, and "very small" in most of the remainder. Of the 39 cases of "permanent" albuminuria, disease of the kidney was either proved to exist, or rendered

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[1] Union Médicale, 1863, and Bulletins de la Société Médicale d'Emulation, Nouv. Sér. t. i., 1864, i., p. 182.

[2] Archives générales, 1864, i., p. 402.

[3] "On the Composition of the Urine," Lond., 1860, p. 186.

highly probable by other symptoms, in 32; and as 3 other cases are excluded because nothing was decidedly known as to the state of the kidneys, permanent albuminuria indicated renal disease in 32 cases out of 36; and if the cases of heart-disease were eliminated, it indicated renal disease invariably.

2. *The greater the quantity of albumen the greater the probability of renal disease:* a large quantity is rarely found except in undoubted acute or chronic Bright's disease. In considering the amount of albumen discharged it is necessary to regard not only the proportion in the particular specimen under examination, but also the total quantity of albumen discharged in 24 hours. Thus a urine may be only slightly albuminous, but if the daily quantity amounts to four or five pints, the total loss of albumen will be very considerable, and the existence of renal disease strongly indicated. There is, indeed, no urine which more surely indicates the existence of Bright's disease than a pale, dilute, abundant urine, containing more or less albumen; on the other hand, it may be looked upon as a rule with very few exceptions that a slightly albuminous urine which is at the same time dense and high-coloured does not indicate the presence of Bright's disease, and that the albumen in such cases is due either to a pyrexial state, or to some impediment to the circulation of the blood.

The microscopic examination of the deposit in the urine (if there be any) also furnishes important information. The deposits which indicate most strongly the existence of organic renal disease are those containing large numbers of tube-casts and renal epithelia—especially if these objects are dotted with oily globules. The deposits which are least indicative of primary renal disease of serious import are those which contain only blood-casts, or a very few small transparent casts.

3. When the urine is found permanently albuminous, and there is no pyrexia, or any other recognisable condition present which can account for the presence of the albumen, the inference is almost irresistible that primary organic disease of the kidneys exists.

It has been stated that it is possible to distinguish between incidental (nervous or idiopathic) albuminuria and albuminuria due to renal disease, by the fact that when certain odorous and pigmentary substances are taken internally they make their appearance in the urine as in health if the albuminuria be of the incidental variety, but not if it be due to disease of the kidneys. Dr. Dyce Duckworth's[1] observations do not support this conclusion. He found that (with some exceptions) iodine, santonine, turpentine, and oil of juniper passed through the kidneys, and appeared in the urine of persons affected with undoubted disease of the kidneys.

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[1] St. Barth. Hosp. Reps., iii., 215.

CONGESTION OF THE KIDNEYS.

By William Roberts, M.D., F.R.S., F.R.C.P.

Definition.—An increase in the amount of blood in the vessels of the kidneys giving rise, if sufficiently intense, to the passage of albumen, blood, and even fibrinous casts of the uriniferous tubules with the urine; it may be altogether independent of any existing inflammatory action in the kidneys. Renal congestion is produced either by an undue supply of blood to the kidneys, or by some mechanical obstruction, impeding the return of blood from these organs. If long continued, structural changes are induced in the kidneys, which bear considerable resemblance to certain forms of Bright's disease. Dropsy is not a necessary concomitant of renal congestion; if it exists, it is most probably due to cardiac or pulmonary disease.

General Pathology.—When congestion of the kidneys is produced by undue determination of blood to those organs, it is termed *active;* and when due to impeded circulation, it is known as *passive* congestion. These forms of renal derangement comprise several conditions which have been grouped by Johnson, Frerichs, Griesinger, and other writers among the varieties of Bright's disease; but although these two classes of cases may be anatomically allied to each other, clinically and practically they are widely different; and it would obviously tend to mislead, if we unite under the heading of "Bright's disease" cases which, as we shall see, present such important differences in their symptoms and general clinical history.

To show that simple hyperæmia of the kidneys, without inflammation, will determine the appearance in the urine of albumen and blood, and even of fibrinous casts, we may briefly review the experiments which have been directed to this point, the first of which were performed by Dr. George Robinson on rabbits. He found that by tightly ligaturing the renal vein, or by making the obstruction incomplete, so that a certain amount of blood was still allowed to circulate through the kidneys, the urine invariably became more or less albuminous, and in most cases bloody. It was found, moreover, that the kidney whose vein was obstructed in this manner was heavier than its uninjured fellow in a proportion varying from 1½ : 1

to 3 : 1.[1] These experiments were repeated by Frerichs with exactly similar results in dogs, rabbits, a cat, and a frog. He also detected casts of tubes in the urine in four out of ten experiments, and in one he observed the presence of renal epithelium.[2]

The rationale of these experiments is simple: the blood accumulating behind the obstruction causes increased lateral pressure upon the walls of the renal vein and its branches. This increased tension is transmitted backwards to the renal capillaries, which in turn become engorged, and their walls are thereby attenuated—a condition highly favourable to the transudation of the serous constituents of the blood through their coats. If the tension be carried to a still farther extent, actual rupture of the capillaries takes place, and blood corpuscles as well as albumen escape with the urine. The Malpighian tufts, from their anatomical arrangement, afford facilities for the ready passage of blood and serum into the urine, and it is thereby rendered probable that in them the transudation process begins, and goes on with greatest activity.

Robinson found that increased *arterial* tension does not so readily determine the passage of albumen and blood into the urine; and his conclusions have been confirmed by Frerichs and Meyer. Endeavouring to test the effect of increased arterial pressure on the urine, Robinson removed one kidney, thinking that the physiological determination of blood to the other might be sufficient to produce albuminuria; but although the experiment was performed five times, in only one instance was the urine albuminous. He then tied the abdominal aorta below the origin of the renal arteries in two weak rabbits, but albumen only appeared in the urine of one.[3] He next removed (to quote his seventh experiment) the left kidney of a middle-sized rabbit; it weighed fifty-four grains. The aorta was then tied below the origin of the renal arteries, and the animal killed two hours afterwards; the right kidney weighed eighty-five grains, and contained six or seven ecchymoses of various extent. The bladder contained about a drachm of bloody and albuminous urine, and this was the invariable result of similar experiments.

Hermann[4] and Overbeck[5] induced artificial albuminuria by temporarily obstructing the circulation in various ways. Thus Hermann tied the renal arteries for a short time, and found that the urine secreted after the ligature was removed was invariably albuminous. Overbeck, in one set of experiments, inflated a bladder previously introduced flaccid into the heart, and allowed it to remain for about a minute; in a second set, he produced asphyxia and consequent arrest of the blood-current by compressing the trachea for four

[1] Med. Chir. Trans. 1843, p. 51.

[2] Die Bright'sche Nierenkrankheit, 1851.

[3] Frerichs states that he could only find traces of albumen in a few cases after such an operation. Meyer, however, saw abundant albuminuria follow this procedure.

[4] Sitzungsberichte der mathem. naturw. Classe der kais. Akad. Vienna, 1861, p. 25.

[5] Ueber den Eiweissharn, ibid., Feb. 1863.

minutes: in both series of experiments, the urine which first flowed after the re-establishment of the circulation was always albuminous and frequently bloody. The albuminuria generally persisted a few hours and then passed away. When shedding of the renal epithelium occurred, it always *followed* the appearance of the albumen, showing that this could not have produced the albuminuria. The probable explanation of these results is, that the Malpighian tufts became engorged in consequence of the temporary interruption of the blood-current, the re-establishment of which produced increased pressure in the renal capillaries, *i.e.*, it produced active congestion of sufficient intensity to cause albumen and blood to appear in the urine.

Results of Renal Congestion.—The inevitable result of persistent and long-continued impediment to the renal circulation is the induction of grave structural changes in the kidneys; the continuance of hyperæmia must of necessity modify the nutrition of the gland-elements, while the presence of blood corpuscles and fibrinous plugs in the delicate tubuli cannot fail to induce eventually more or less extensive destruction of those structures. It would also appear that an excessive production of adventitious connective tissue, leading ultimately to contraction and atrophy, is one of the most important results of long continued congestion of the kidneys.

(A.)—ACTIVE CONGESTION.

SYNONYM.—Catarrhal Nephritis (Virchow, Rosenstein).

ETIOLOGY AND PATHOLOGY.—The kidneys participate in the general hyperæmia of the internal organs which occurs in the course of eruptive and continued fevers, of croup, diphtheria, cholera, erysipelas, pyæmia, acute rheumatism, pneumonia, and other inflammatory diseases; they are not unfrequently however the seat of a disproportionate determination of blood, leading to the presence of albumen in the urine. As a rule, there is a mere trace of albumen found in these cases, which diminishes *pari passu* with defervescence, and in a few days totally disappears; but sometimes it is more abundant and accompanied with a few blood-corpuscles, transparent tube-casts, and scattered renal epithelium: tenderness in the loins is sometimes present.

This condition is distinguished from genuine Bright's disease, which may occur in connection with the same pyrexial maladies, by the absence of anasarca, by the undiminished urea-excretion, and by the period of its occurrence: thus albuminuria from congestion coincides with the height of the fever, and subsides therewith; genuine Bright's disease, on the contrary, occurs as a sequela, towards the close of the pyrexial stage or at the beginning of convalescence.

The pathological appearances when death has taken place from the primary fever during active renal congestion are the following: the organs are enlarged and engorged with blood and present minute ecchymotic patches throughout their substance; Virchow has pointed out the existence in these cases of a catarrhal condition of the canals of the pyramids characterised by the detachment and disintegration of the epithelium of the straight tubes: the epithelium sometimes undergoes fatty metamorphosis.

The frequency of this complication of zymotic diseases varies in different epidemics. During a severe typhus epidemic in 1857, Rosenstein found that most of the patients had transient albuminuria with tube-casts, unattended, however, with serious consequences: in the sporadic typhoid of Manchester, albuminuria is decidedly rare.

Another not very common, perhaps (as suggested by Rosenstein) because often-overlooked cause of active renal congestion, is simple exposure to cold, quite independently of any specific fever. If the urine does not happen to be examined, the case will probably be considered a simple febricula, the symptoms of the two affections being very similar.

Certain irritants act as special stimuli of the urinary organs, and excite hæmorrhage from the kidneys and lower urinary passages. Among these may be enumerated cantharides, turpentine, cubebs, copaiba, nitrate of potash, and also, in very rare cases, quinine and arsenic. Bouillaud found that when large blisters were applied to scarified portions of the skin, albumen almost invariably appeared in the urine, ceasing generally in two or three days: in a few cases it persisted for weeks. After death, the kidneys were found strongly congested and studded with minute ecchymoses, the mucous membrane of the pelvis and ureters, sometimes also of the bladder, injected, and covered here and there with false membranes.

Dr. Johnson records a case in which a few hours after the administration of half an ounce of turpentine the urine was bloody and contained "blood-casts," with a few small inflammatory cells, but no epithelium. On the sixteenth day, the urine was free from albumen and blood.

The writer has known one person in whom quinine invariably produced hæmaturia; two similar cases are cited in the *British Medical Journal*, vol. i. 1870, p. 11. This effect is probably due to idiosyncrasy.

Two cases of poisoning by sulphuric acid are related by Leyden and Munk, in which the urine contained albumen and tube-casts.[1]

Frerichs includes irritants of this class among the exciting causes of genuine Bright's disease, and cites two cases by Reinhardt, in which the abuse of copaiba and cubebs was followed by renal degeneration, which proved fatal in one case; but, as Rosenstein points out, the kidneys were probably diseased prior to the administration of the irritants. There is no sufficient evidence to prove that genuine Bright's disease has really ever followed an overdose of any of these stimulants.

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[1] Archiv. f. path. Anat. Bd. xxii. p 237.

Albumen and even blood not unfrequently appear in the urine in the later stages of diabetes; the excessive action of the kidneys in this disease keeps up a constant congestion of these organs, and permanent structural changes ultimately take place—degeneration of the epithelium, development of cysts, and other morbid changes—which are classified with genuine Bright's disease.

In rare instances, active renal congestion causing albuminuria seems to be attributable to the compensatory hypertrophy of the left ventricle which follows aortic regurgitant disease. In this condition the propulsion of blood into the aorta when the orifice is patulous, takes place with very great force, and the arterial tension at the close of the ventricular systole, rises considerably above the normal maximum, though it may be counterbalanced or even more than counterbalanced by undue diminution of tension during the ventricular diastole. In practice, this form of congestion seems to be rare, however; the writer has repeatedly examined the urine of persons with immense enlargement of the left heart, and in only three or four instances has he found albumen in the urine. In a case recorded by him,[1] in which there was enormous hypertrophy of the left ventricle, it was distinctly observed that the proportion of albumen oscillated with the activity of the heart; when the ventricle was in high action, the albumen increased, and when it became more quiescent under treatment the albumen almost disappeared.

(B.)—Passive Congestion.

Etiology and Pathology.—That this form of renal derangement is produced by an impediment or obstruction to the return of blood from the kidneys is clearly shown by the results of the experiments of Robinson, Frerichs, and others already cited. Two degrees of this obstruction are encountered clinically; if sufficiently intense, albumen and blood appear in the urine, but if insignificant, the urine is not rendered albuminous but becomes scanty, high-coloured, dense, and prone to deposit lithates copiously. These changes in the characters of the urine are not always proportionate to the amount of obstruction to the circulation; for cases are occasionally met with in which the urine contains not even a trace of albumen while the symptoms of intense venous congestion, dropsy, orthopnœa, and pulsating jugulars are present. In other cases, the urine may be albuminous and bloody, while the general symptoms of venous obstruction are by no means strongly marked.

The obstruction may be seated in the chest or in the abdomen; if in the chest, it may be due to valvular heart disease, to emphysema, or to pleuritic effusion: if in the abdomen, it may be produced by the pressure of a gravid uterus, a cirrhotic liver, or other tumour on the upper course of the inferior vena cava, or upon the emulgent veins.

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[1] Op. cit. p. 344.

Robinson in the course of his experiments found that vigorous animals exhibited the urine-changes after ligature of the renal veins in much greater intensity than weakly animals: this difference he attributed to the fact that in strong animals the powerful contractions of the ventricle served to maintain a greater counter-pressure on the arterial side of the renal circulation, and so intensified the intra-renal pressure or congestion. That this holds good clinically as well as experimentally is well illustrated by the case of a woman, aged forty-two, suffering from excessive and universal emphysema, observed by the writer. In this case, the albumen, which amounted to one-third when she first came under observation, gradually disappeared, although the intra-thoracic obstruction gradually increased, and eventually produced death by asphyxia. This was explained by the diminishing pressure in the arterial system from gradual failure of the heart's power, in consequence of the inability to take food, which diminished the mass of the blood, and the increasing blood-poisoning from defective aeration, which gradually depressed and ultimately destroyed the contractility of the ventricle.

The state of the kidneys in passive congestion varies with the duration of the obstruction. If recently established, the kidneys are found simply enlarged and engorged, as in the case of the rabbits whose renal veins were tied by Robinson. But when the obstruction has lasted for months or years, certain alterations in the renal structure take place. The following description of the kidneys in old-standing passive renal congestion is based on a comparison of several accurate examinations. The organs are uniformly reddened and are decidedly smaller and harder than natural; their surface is generally smooth, but sometimes granular, the proportion of cortical to pyramidal substance is not much altered; in very chronic cases, the cortex may be somewhat atrophied. The epithelium of the convoluted tubes is irregular, sometimes fatty, and frequently contains pigment. The straight tubes are often dilated and varicose, being filled with granular, opaque epithelium. The basement membrane of the canals is thickened, and the venous radicles are greatly dilated. The Malpighian bodies are highly injected but otherwise natural, except in very protracted cases, when they become a little atrophied, and thickening of their capsules takes place. The inter-tubular connective tissue is increased in quantity, especially in the pyramidal portions.

However great may be the difficulty in separating these cases from chronic Bright's disease anatomically, the different course of passive renal congestion from that of Bright's disease of independent origin, and of Bright's disease coming on in the course of chronic bone-disease or phthisis, renders their clinical differentiation very simple. In passive congestion, the renal derangement oscillates *pari passu* with the rising and falling intensity of the venous obstruction, making no independent progress, but remaining throughout a subsidiary complication of the primary affection, and developing none of the special characteristics of Bright's disease, as uræmia and the like. When renal disease declares itself in the course of chronic phthisis, its course forms a complete contrast to that of congestion. It at once assumes a formidable position, changing the entire clinical aspect of the case; the pulmonary affection sometimes even undergoes a retrograde process, and is as it were supplanted by the more rapidly fatal renal disorder.

TREATMENT OF ACTIVE AND PASSIVE RENAL CONGESTION.

Congestion of the kidneys, whether active or passive, does not often call for separate treatment, as its course and intensity are usually contingent on the progress of the primary disorder of which it is a secondary phenomenon. Sometimes, however, active congestion is of independent origin, or, although secondary, it may be sufficiently threatening to demand special attention. The most efficacious agents in the treatment of active renal congestion are perfect rest of the body, cupping the loins, brisk purgatives, the warm bath, and other diaphoretics.

Passive congestion from cardiac and pulmonary obstructions can be most efficiently relieved by the use of appropriate remedies for the primary ailments; but when it is due to the pressure of a pregnant uterus, it not unfrequently claims energetic treatment on its own account. Cupping can only be of service when the congestion is due to a temporary cause, such as pregnancy, and here it is sometimes of signal benefit. In the more ordinary forms, the application of gentle counter-irritants will be found more serviceable; at the same time, derivation by the bowels and skin will materially aid in relieving the over-loaded organs.

BRIGHT'S DISEASE.

By William Roberts, M.D., F.R.S., F.R.C.P.

Definition.—A generic term embracing certain types of acute and chronic renal disease, characterised by albuminuria and dropsy, and frequently also by the supervention of various secondary conditions, due to a deteriorated condition of the blood.

Synonyms.—Albuminuria; Morbus Brightii (Lat.); Maladie de Bright (Fr.); Die Bright'sche Nierenkrankheit (Germ.); Malattia di Bright (Ital.).

History, Classification.—Although a connection had been previously shown by Dr. Blackall to exist between dropsy and albuminuria, Dr. Bright was the first to establish the co-existence of these symptoms with certain morbid conditions of the kidney by the publication of his "Report of Medical Cases" in 1827. His researches on this subject were followed up by those of other observers, notably by those of Sir Robert Christison; and about this time considerable impetus was given to the study of renal diseases by the researches of Mr. Bowman on the minute anatomy of the kidney. It would obviously lead us too far to give an account of all the work subsequently done in this field; suffice it to say that the histological researches of Busk, Toynbee, and Simon in this country, and of Henle, Rokitansky, and Virchow in Germany—and the clinical observations of Johnson, Frerichs, Basham, Dickinson, Grainger Stewart, and many others, have combined to elucidate the subject by throwing much light on its pathology, and by furnishing us with important data for prognosis and treatment. The labours of Dr. George Johnson, more especially in the microscopic examination of the organic admixtures of the urine—renal epithelium and casts of the uriniferous tubes—have yielded results of the highest clinical value, and entitle him to a pre-eminent notice in this field of pathology.

The varieties of Bright's disease, while presenting considerable diversity not only in the acuteness of their course, but also in their modes of origin and in their symptoms, yet have points of resemblance so strong and so numerous, that they form an easily recognised clinical group. This resemblance is due in great measure to the circumstance that the structural changes in the kidneys, however varied, produce the same ultimate results, namely, impoverishment of the blood from loss of albumen, with blood-poisoning from retention within the body of the excrementitious matters of the urine; and the more prominent symptoms in Bright's disease arise from this changed composition of the blood, rather than from the *direct* effect of the organic changes in the kidneys.

Numerous attempts have been made to classify the varied conditions of the kidney found after death from Bright's disease and to associate each with its appropriate clinical history; hitherto, however, no classification has received general assent, and it is to be regretted that the result of these attempts has chiefly been to add a confusing nomenclature to the inherent difficulties of the subject. Opinions are divided, in the first place, as to whether a fundamental unity underlies the apparent diversity, *i.e.*, whether the "large, smooth, white kidney," the "small smooth kidney," the "granular uncontracted kidney," and the "granular contracted kidney," are successive stages of one and the same pathological process, or represent essentially different diseases. The distinguished physician of Guy's Hospital, whose name has been given to this group of diseased conditions, was by no means certain that his conjecture that there were three different forms of disease was a correct one; "on the contrary," he remarks, "it may be that the first form of degeneracy to which I refer never goes much beyond the first stage; and that all the other cases, together with the second series and the third, are to be considered only as modifications, and more or less advanced states of one and the same disease."[1] Notwithstanding all the work which has been done in the field opened up by Bright, the doubts expressed by him are not yet wholly cleared up.

Frerichs considers that Bright's disease is essentially *one*, and that it is of an inflammatory nature. He recognises three forms of anatomical change in the kidney, which he regards as progressive stages of the same pathological process, namely :—

1. The stage of hyperæmia and commencing exudation.
2. The stage of exudation and commencing change of exudation.
3. The stage of degeneration and atrophy.[2]

Dr. Johnson, on the other hand, recognises several distinct processes under the generic heading of Bright's disease. There are two principal varieties according to him, both of which are of an inflammatory nature: the one is characterised by shedding and more or less complete disintegration of the epithelial lining of the uriniferous tubes, and was named by him *desquamative nephritis;* in the other, no such desquamation takes place, but the intertubular structures are the portions of the organ affected, constituting his *non-desquamative nephritis.* Besides these, he assigns a separate place to "fatty degeneration" and to "waxy degeneration" of the kidney.[1]

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[1] Report of Medical Cases, by Richard Bright. Lond. 1827, p. 69.

[2] Die Bright'sche Nierenkrankheit. Braunschw. 1851.

Dr. Dickinson, in his treatise,[2] divides Bright's disease into three main groups:—

1. Tubal nephritis—in which the uriniferous tubes are the seat of inflammatory action.
2. Granular degeneration—in which there is increase and subsequent contraction of the intertubular matrix of the kidney.
3. Depurative disease—synonymous with what is known as amyloid, waxy, or lardaceous infiltration of the kidneys.[3]

Dr. Grainger Stewart, in his work on "Bright's Diseases,"[4] a term which he regards as more truly descriptive than "Bright's disease," adopts the nosological arrangement of Virchow with certain modifications. He classifies the cases in the following manner:—

1. The inflammatory form—of which there are three stages:—
    - (*a.*) That of inflammation.
    - (*b.*) That of fatty transformation.
    - (*c.*) That of atrophy.
2. The waxy or amyloid form—of which also there are three stages:—
    - (*a.*) That of degeneration of the vessels.
    - (*b.*) That of secondary changes in the tubes.
    - (*c.*) That of atrophy.
3. The cirrhotic, contracting, or gouty form.

In addition to these, he describes two mixed types, in which the waxy and cirrhotic forms are respectively combined with the inflammatory form.

Without entering into a discussion of the merits of these and of many other classifications which have been propounded, the writer would merely indicate that in the following pages the classification adopted is based on clinical, in preference to anatomical, characteristics. Two main divisions are therefore made—Acute and Chronic Bright's disease. The former embraces a compact and universally recognised group formerly known as "inflammatory dropsy;" it corresponds to the first stage of Frerichs, to the acute desquamative nephritis of Johnson, and to the acute tubal nephritis of Dickinson. The latter division includes the protracted cases, which have either passed from the acute form into a chronic state, or, which is far more frequent, have been chronic from the first. Three types of chronic Bright's disease are recognised:—

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[1] On Diseases of the Kidneys. Lond. 1852; and Med. Chir. Trans. vol. xlii.

[2] On the Pathology and Diseases of the Kidney, Part II. Lond. 1877.

[3] In his recently issued second edition, Dr. Dickinson gives up the term "depurative" and adopts the old term "lardaceous."

[4] A Practical Treatise on Bright's Diseases of the Kidneys. 2nd Edit. Lond. 1871.

1. Cases which have lapsed from the acute state (kidney smooth, white, generally large, exceptionally dwindled).
2. Cases which have been chronic from the beginning (kidney granular, red, contracting).
3. Cases associated with waxy, lardaceous, or so-called amyloid degeneration of the kidneys.

The presence of fat in the renal substance and in the epithelium of the tubes not being limited to any special type of renal degeneration has no claim to a separate consideration.

GENERAL ETIOLOGY OF BRIGHT'S DISEASE.

It will be convenient to consider some points bearing on the etiology of Bright's disease as a whole, leaving the special etiology of each type to be considered separately under its own heading. To obtain an idea of the *prevalence* of Bright's disease, some inferences may be deduced from the returns of the Registrar-General, the value of which is, however, greatly diminished by the want of uniformity in our nomenclature. Cases registered as deaths from Bright's disease are entered in these returns as "nephria," but it is evident that the larger number even of the cases of Bright's disease recognised as such during life, are not so registered, but are classified under "nephritis" and "kidney disease." During 1874, 3,342 deaths were registered in England from "nephria," 864 from "nephritis," and 2,999 from "kidney disease," making a total of 7,205. This yields a proportion of only 1·4 per cent. of the total deaths from all causes—a number probably considerably below the true proportionate mortality from Bright's disease; in all likelihood, however, a large number are overlooked in these returns and are to be found among the 4,688 entered under "dropsy," or among those entered under "convulsions," "pneumonia," and other headings.

Age and Sex.—Bright's disease is nearly one-fourth more common in men than women, the proportion being 1,936 males to 1,406 females. This excess of deaths among males obtains at every age, but is not equal at the different periods of life, being most marked between forty-five and sixty-five. The mortality from Bright's disease shows a progressive increase from childhood up to about the age of fifty; during the next twenty years the mortality continues steady at a somewhat lower rate; but during the succeeding decade a decided diminution in the mortality from Bright's disease takes place, although at this epoch the general mortality is at its highest point. These facts are exhibited in the following table:—

Table showing the number of Deaths registered as "Nephria" (Bright's Disease) in England in 1874 at the different periods of life.

	Under 5 yrs.	5–15 yrs.	15–25 yrs.	25–35 yrs,	35–45 yrs.	45–55 yrs.	55–65 yrs.	65–75 yrs.	75 yrs. & upw.	Total at all ages.
Males	89	79	116	220	332	398	359	240	89	1936
Females	54	72	107	216	239	229	234	193	60	1406
Both Sexes ...	143	151	223	436	571	627	593	433	149	3342

Cold.—The complex series of impressions comprised in the familiar phrase "taking cold," is a very common cause of Bright's disease in its acute form, while the more slow and continuous operation of cold is a fruitful source of chronic Bright's disease. Hence persons whose occupation exposes them to cold and damp, and the inclemency of weather — those who work in hot workshops, and are liable to sudden vicissitudes of temperature — the indigent classes living in damp cellars, ill-fed, and insufficiently clothed—all these are specially liable to become the victims of Bright's disease.

Various theories of the *modus operandi* of cold in inducing renal disease have been advanced. It has been supposed that the mere diversion of increased quantities of fluid as in checked cutaneous excretion is sufficient to inflame the kidneys; but it is beyond question that when much fluid is taken into the system the kidneys may be caused to excrete many times the natural amount of urine and no inflammation result. Dr. Johnson contends that the defective action of the skin causes certain deleterious matters to accumulate in the blood, and that the burden of their elimination is thrown upon the kidneys to their injury; but it must be remembered that suppressed cutaneous transpiration ushers in a multitude of inflammatory and febrile conditions without inducing renal disease: it is impossible to predicate—when a person has "taken cold"—what organ will be affected—whether the pleura, the lung-tissue, the bronchial tubes, or the kidneys, or some other organ or part of the body. No *special* relation between suppressed cutaneous secretion and the genesis of renal disease can therefore be said to exist.

Intemperance.—The abuse of spirituous liquors is a prominent determining cause of Bright's disease. Sir R. Christison estimates the proportion of cases due to this cause in Edinburgh as three-fourths or four-fifths of the total number. He observes that dram-drinkers who regularly take ardent spirits several times a day short of intoxication are liable to renal disease as well as habitual drunkards. Dr. Dickinson has devoted a chapter in his recent work[1] to the consideration of alcohol as a cause of renal disease. From pathological observations made on the bodies of persons who had died of delirium tremens, or from the abuse of spirituous drinks, and from a want of correspondence between the death-rate from Bright's disease as shown by the Reports of the Registrar-General and the degree of intemperance prevailing in different localities, he concludes that the use of alcoholic drinks is comparatively inoperative as causing disease of the kidneys. The writer has elsewhere[1] examined these data, and has shown, he believes, conclusively that the arguments advanced by Dr. Dickinson are inadequate to disturb the old and almost universal opinion respecting the injurious influence of alcohol on the kidneys.

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[1] Op. cit. p. 600.

Malt liquors, though less pernicious than spirits, are also influential in the production of Bright's disease, if freely indulged in; and as intemperate habits frequently accompany personal uncleanliness and exposed occupations, labourers, cabmen, carters, hawkers, and persons under similar circumstances, form an undue proportion of the victims of Bright's disease.

Constitutional Vice.—Many of the chronic cases are associated with tuberculosis or struma and chronic plumbism; while gout and constitutional syphilis are by no means infrequent antecedents among the wealthier classes.

Ague.—The writer has not met with a case occurring sequential to ague. Becquerel and Frerichs also have never found evidence of kidney-disease in dropsies following intermittent fevers. Rosenstein, however, found that 23 per cent. of the cases of Bright's disease in the Dantzic Hospital were referable to antecedent ague. The frequency of this complication is influenced by the character of the epidemic as shown by Heidenhain; he found that neither dropsy nor renal mischief occurred in the earlier epidemics observed by him in Marienwerder, but in the latest epidemic secondary renal disease followed in almost every case.

Pregnancy.—While women who are the subjects of Bright's disease may become pregnant, and pregnant women are liable, like other people, to contract Bright's disease from any of its ordinary causes, there is a third category in which pregnancy has really caused Bright's disease. The Registrar-General's reports furnish some valuable evidence on this point. In the five years between 1857 and 1861, 6,220 deaths were registered from Bright's disease. Of these 3,699 were males and 2,521 females—the relative proportion of the sexes at all ages being sixty-eight females to every hundred males; but during the child-bearing period (from twenty to forty-five), the proportions were eighty women to every hundred men, while after the age of forty-five the proportionate mortality from Bright's disease fell to fifty-nine women for every hundred men. The only conclusion that can be drawn from these statistics seems to be that pregnancy is a fruitful cause of Bright's disease.

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[1] Brit. Med. Journ. vol. ii. 1871, p. 521.

Chronic affections of the lower Urinary passages.—The foundations of renal disease are frequently laid by chronic cystitis, long-standing stricture of the urethra, and other local affections. The etiology of these cases is discussed in a separate article (Secondary Diseases of the Kidney).

A large number of cases of chronic Bright's disease occur in which no exciting cause can be detected; in some of these the renal disorder is only a part of some wide-spread cachexy, such as atheromatous or lardaceous degeneration.

Wells and Blackall believed that mercury was capable of producing albuminuria and renal mischief; but Rayer and Desir out of forty cases treated with mercury at the Hôpital des Vénériens only found a slight quantity of albumen in two, in both of which the existence of pus accounted for its presence. Rayer also states that for years he had used mercurial preparations therapeutically, and had also treated a large number of gilders with mercurial trembling, without having observed a single case of dropsy with coagulable urine.

(A.)—ACUTE BRIGHT'S DISEASE.

SYNONYMS.—Inflammatory Dropsy; Acute Diffuse Nephritis; Acute Desquamative Nephritis (Johnson); Acute Tubal Nephritis (Dickinson).

MORBID ANATOMY.—The kidneys are always enlarged, sometimes to twice their normal size; their surface is smooth and generally of a deep, dusky-red colour, but sometimes a pale fawn, or it may be mottled red and white. The capsule is thin, transparent, and easily stripped off; the superficial veins are larger and more distinct than natural. On section, the red congested kidney exudes abundantly a bloody sanies from the cut surface, which is of a dusky red hue, and studded with minute darker red points—the engorged Malpighian corpuscles; a number of hæmorrhagic spots may usually be seen scattered through the cortex or beneath the capsules. The cortical substance is found relatively much increased. The pale and the mottled kidneys present a contrast of colour between the cortex and the pyramids. The latter appear unnaturally red, and radiating lines of red spread, fan-like, from their bases into the cortical substance which is smooth, white or yellowish white, and spotted like ivory.

Under the microscope the chief and characteristic alteration is found to be an immense increase of the epithelium, mixed with more or less blood, choking up the lumen of the convoluted tubules, whose diameters, in extreme examples, are increased to twice or even thrice their normal size. The individual cells may be natural; more commonly they are granular, opaque, or disintegrated; they sometimes contain a few oily particles. The straight tubes afford evidence of the same process but in a less intense degree, for their larger bore and direct course favour the escape of the detached epithelium, so that some of them are partially or wholly denuded. Fibrinous exudation is found in the tubes of the cortical and pyramidal portions in the form of transparent cylinders of various size, according as the tubes have preserved or have shed their proper epithelial lining. The intertubular matrix is unaffected, and the renal capillaries and Malpighian tufts are either natural or intensely injected.

PATHOLOGY.—The disorder is essentially a catarrh of the uriniferous tubes with enormously increased epithelial growth. The first step in the morbid process is an inflammatory congestion of the organs with rapid swelling, and more or less extensive rupture of the capillaries, especially those of the Malpighian tufts; then increased production of epithelial cells, packing up and distending the tubules, and thereby compressing and impeding the circulation through the renal capillaries. The depurating functions of the organs are thus necessarily impeded and the blood is poisoned with excrementitious matter; the urine becomes scanty and deficient in its proper constituents, and as it percolates through the diseased ducts it detaches whole tracts of their epithelial lining or carries with it loose epithelium, blood, and fibrinous exudation, which forms the grumous sediment seen in the urine in these cases. The pale or mottled appearance of the kidneys sometimes observed is due to the extreme proliferation of the epithelium, which overpowers the natural red colour, rather than to a positive deficiency of blood in the organs. The writer has seen this condition attain an extreme degree in six weeks: Dr. Dickinson states that it may occur within four days.

COURSE AND SYMPTOMS.—The disease is usually traceable to some definite exposure to cold or a fit of intemperance, or it occurs as a sequela of scarlet fever or (very rarely) of some other zymotic disease. It is generally ushered in abruptly with the usual symptoms of pyrexia—chilliness or rigors, soon followed by febrile reaction, headache, nausea, vomiting, dryness of skin, and dull pain and tenderness in the loins. Dropsy is an early symptom: the face first becomes pale and puffy, thus acquiring a heavy, stupid expression; this is followed by general anasarca, which is resistant on pressure, and pits very slightly, if at all, and by effusion into one or more of the serous cavities.

Urine.—There is a frequent desire to urinate, especially when the patient is recumbent. The quantity of urine in the earlier periods is greatly diminished, in extreme cases it may be reduced to four or six ounces in the twenty-four hours, or it may be even totally suppressed for two or three days. It is usually of a smoky or dusky hue—in some cases dark-brown like porter; its natural odour is replaced by a faint, unpleasant smell like that of "flesh-washings." It is commonly acid; rarely alkaline from fixed alkali. Its specific gravity ranges between 1,020 and 1,030; in one instance which occurred to me, it was 1,065. It is highly albuminous, sometimes becoming quite solid on boiling. On standing it deposits a copious, flocculent, chocolate-coloured sediment which, on microscopic examination, is found to consist of blood-corpuscles, loose renal epithelium and free nuclei, tube-casts, shapeless masses of coagulated fibrine, and the broken debris of all these structures, besides epithelium from the renal pelvis and the bladder. (See Fig. 1). The tube-casts are abundant and varied: the most common are of "medium" size, transparent, and studded with epithelium or blood-disks; along with these may be some "very large" and some "very small" hyaline casts, together with opaque granular casts. Rarely, a few small specks of oil may be seen either on the casts or within the epithelia, in the early stages of the disease. The quantity of albumen in the urine varies from eighty to about 400 grains per day; the proportion per 1,000 parts during the height of the disease has been variously estimated by Frerichs as ranging between 8·2 and 24·8, by Christison as 27, and by Heller as 57. The natural solid constituents of the urine are diminished according to the obstruction in the kidneys: the excretion of urea falls from 400 or 500 grains to 100 or 200 grains, and the inorganic salts are considerably reduced, while uric acid maintains about its usual quantity.

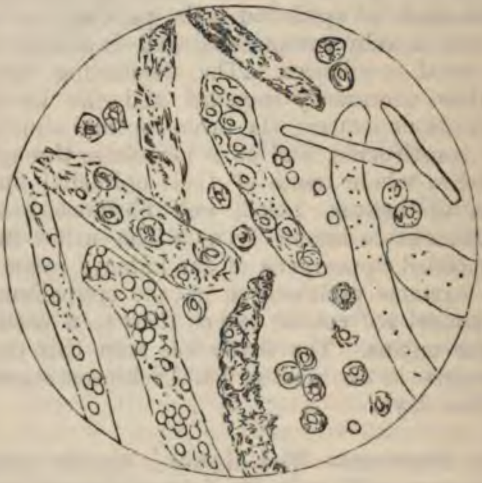

FIG. 1.—Transparent, granular, blood and epithelial casts from a case of acute Bright's disease; free renal epithelium; and blood disks.

Blood.—Owing to the unnatural drain through the kidneys, the blood becomes poorer in albumen and more watery, while undue accumulation in it of urea, uric acid, and extractives takes place. As the disease advances, general anæmia is produced by the diminution of the blood-disks. Fibrine is usually in excess and the blood exhibits a buffy coat: fat and inorganic salts retain their normal ratio. Frerichs gives the three following analyses of the blood in the early period of acute Bright's disease:—

	I.	II.	III.
Specific Gravity	1025	1022	1019
1,000 parts of serum contained:—			
Water	908·10	915·88	938·9
Solids	91·90	84·12	61·1
Albumen	81·40	72·00	51·7
Fat	1·42	1·53	9·4
Extractives and Salts	9·09	10·59	

TERMINATIONS.—After a period varying from a few days to some weeks, the disease proceeds to one of three terminations: it may end in recovery, in death, or by lapsing into the chronic state.

In the most favourable cases the urine first improves; its quantity increases to three or four pints daily; its density falls below the natural mean; the blood, tube-casts, and albumen gradually disappear from it, and *pari passu* with the changes in the urine, the skin becomes moist, and the serous effusions are re-absorbed. The rate of progress varies extremely: if the urine is perfectly free from albumen within six weeks or two months, recovery may be regarded as rapid. The shortest period that I have known to elapse between the earliest symptoms and the complete re-establishment of the normal state, was ten days.

But in other cases convalescence is protracted over eight, twelve, or eighteen months, during which, the urine continues abundant, of low density, and often of a pink colour from slight admixture of blood; the anasarca may recur and disappear several times, accompanied with slight febrile exacerbations. In one such case observed by me the symptoms finally subsided in five months, the urine being perfectly free from albumen ten months later; and in a second case, the urine contained a slight admixture of blood for more than a year. In these two cases, and in a third of similar nature, the urine was of uniform character: it was copious (three or four pints daily), of low density, slightly mixed with blood, and slightly albuminous; the renal derivatives were devoid of fat, and comparatively scanty throughout the convalescence. It sometimes happens, however, that during recovery from acute Bright's disease, the renal epithelium and casts are found slightly fatty, and if the case should come under observation for the first time at this stage it may simulate chronic Bright's disease and prove very embarrassing for the prognosis.

There may be certain deviations from the usual course and symptoms of the disease; thus, although anasarca commonly appears first in the face, under the eyes, and afterwards invades the trunk and extremities, it may show itself first in the feet, hands, or scrotum; or œdema may appear in all parts of the body simultaneously. The effusion may also shift its seat from time to time, or it may induce the sudden accession of alarming or fatal symptoms from its being poured out in disproportionate abundance in certain localities, such as the lung, pleura, and submucous tissue of the glottis. Moreover, though the anasarca usually disappears some days or weeks or even many months before the urine is free from albumen, the converse sometimes obtains, especially in individuals of lax frame and anæmic tendency; such cases are apt to mislead if seen for the first time, after the albumen has disappeared from the urine,—careful inquiry into the patient's previous history will elicit their true nature.

COMPLICATIONS.—These are chiefly comprised under two groups, viz.: secondary inflammations of the serous membranes and the lungs, and a series of nervous phenomena known under the name of uræmia.

Secondary inflammations are much more common in the later stages of chronic Bright's disease than in the acute form: of these, pericarditis, although rare, is the most certainly fatal. Pneumonia is more common and is also frequently the immediate cause of a rapidly fatal issue. Pleurisy and peritonitis, though less lethal, are not unfrequent, while bronchitis is almost invariably present. Should the anasarca become excessive there may be inflammation or even mortification of the integuments of the legs.

The uræmic phenomena have been attributed to the retention in the blood of the excrementitious matters of the urine; they usually follow an excessive diminution or suppression of the urine from increasing obstruction in the kidneys. They consist in a train of nervous symptoms—headache, vomiting, diarrhœa, convulsions, coma —the consideration of which will be postponed till we come to discuss uræmia in connection with Bright's disease generally.

ETIOLOGY.—Acute Bright's disease, though not absolutely confined to any age, occurs in the immense majority of cases during childhood and youth. As a rule, the persons attacked have previously been in good health; in two instances, however, I have seen the disease complicated with acute pulmonary tuberculosis.

The exciting cause is usually some definite exposure to cold (such as lying or sleeping in a damp bed or on the damp ground, sitting in wet clothes, or in a current of cold air, or drinking cold water when in a state of perspiration) or a fit of intemperance. A large proportion of the cases are sequelæ of scarlet fever or, much more rarely, of some other zymotic disease, while, in some instances, the affection is the result of pregnancy; it may also be caused by deficiency of food, with fatigue and mental anxiety.

DIAGNOSIS.—During the height of the attack the symptoms and the changes in the urine are so unequivocal that the disease can hardly be mistaken for any other. But when the pyrexial stage is past, and the case becomes protracted, there is often great difficulty in determining whether we have to deal with the declining periods of an acute and curable disorder, or with a disease which has lapsed into the chronic and irremediable state, or with a disease which has been chronic throughout. The recent and curable forms are generally distinguished by the presence of blood and renal epithelium in the urine, by the absence of fat in the discharged elements, and by the absence of long-standing complications, such as hypertrophy of the left ventricle, phthisis, caries, necrosis, and joint disease. It is important to consider carefully the previous history of the patient and the ostensible cause of the disorder; the less clearly a case can be traced to a definite exposure to cold, to a fit of drinking, or to scarlet fever or some other zymotic disease, the more reason *pro tanto* is there to fear that confirmed Bright's disease is established.

PROGNOSIS.—The prognosis is generally favourable: a large majority of the cases undoubtedly recover. Frerichs reckons the recoveries as two-thirds of the individuals attacked; but probably this proportion is below the truth if the scarlatinal cases be included; we are still in want of precise data on the point. The prognosis is decidedly more favourable in the aged than in the young; I have several times seen the disease in persons over sixty, and once in a man on the verge of eighty; in most of these it proved mild, and in all eventuated in recovery.

Recovery cannot in any case be considered complete until the urine has become perfectly free from albumen. The anasarca may totally disappear, and blood cease to tinge the urine; the quantity of the secretion may increase considerably, the pyrexia pass away, and the general condition of the patient be greatly improved, but if a considerable amount of albumen continues to appear in the urine, there is great ground for apprehension that the disease is lapsing into a chronic state, or that the amendment is but a temporary lull in the symptoms, to be followed at no distant period by an exacerbation which shall prove more disastrous than the original attack. The worst consequences are to be feared if the urine become progressively scantier, of higher density, and more abundantly charged with albumen, tube-casts, and renal epithelium. The advent of inflammatory complications, of œdema of the lungs or glottis, and, above all, of decided signs of uræmic poisoning, are of equally evil augury, and leave but slender hopes of ultimate survival.

TREATMENT.—The double indication of treatment is to relieve the kidneys and to promote the action of the other excretory organs. If the case is seen at the onset the patient should be at once confined to bed, swathed in flannel, and made to lie between the blankets. The loins should be immediately cupped to eight or twelve ounces if the patient be an adult, to two or three ounces if a child. Abstraction of blood must be cautiously practised however, on account of the tendency to anæmia in the later periods of the attacks. If the patient's health is broken by previous disease or if he is constitutionally weak, even local depletion is better avoided. If severe headache, coma, or convulsions occur, the cupping may be repeated. In threatening cases where the fever runs high, venesection may be practised in sthenic cases. After the abstraction of blood, a large hot linseed-meal poultice should be applied to the loins and renewed every three hours. The action of the skin should be promoted by the use of hot-air or hot-water baths; if no convenience for these exist, the "blanket-bath" forms an excellent substitute. Of medicinal diaphoretics, citrate of potash draughts given in effervescence every two hours, or a mixture of two or three drachm doses of liq. ammon. acet. with fifteen drops of tinct. hyoscyam. in one ounce of infus. lini are very serviceable. Dr. Barlow recommends $\frac{1}{4}$ to $\frac{1}{8}$ gr. of tartar emetic; I have employed it every four hours with the best results. Dr. Johnson speaks highly of antimonial wine, sometimes combined with Dover's powder. The bowels should be freely acted on every other morning by an active purge, such as the compound jalap powder. The use of mercury is objectionable, on account of the extreme susceptibility of patients with Bright's disease to the physiological effects of the drug. Severe salivation sometimes follows very small doses; in one of my patients profuse ptyalism was produced by two grains of blue pill with extract of colocynth taken on two alternate mornings.

Objections have been made, on theoretical grounds, to the use of the saline diuretics (acetate and citrate of potash) in acute Bright's disease;* experience has proved, however, that they may be employed with great advantage. In a considerable number of cases of acute Bright's disease coming under treatment early, I have almost invariably obtained the best results by the free administration of citrate of potash; and in no instance where the urine has been rendered alkaline in the first week of the disorder have I observed the more severe uræmic symptoms or secondary inflammations. This remedy, however, has proved inefficacious in my hands when the fever has entirely subsided while the urine remains bloody and albuminous. Digitalis may be freely employed in any stage. Sir R. Christison recommends one- or two-grain doses of the powder in a pill, or ten to twenty minims of the tincture of digitalis combined with about two drachms of cream of tartar daily in five ounces of water—he considers this combination superior to either remedy given singly. Digitalis may also be applied externally in the form of cloths steeped in the infusion and laid over the abdomen. Dr. Dickinson attaches considerable importance to the free administration of water as a diuretic to facilitate the elimination of the urinary solids by the kidneys and so diminish the risk of uræmia. The diet should consist of light farinaceous substances with milk, beef-tea, and broths;

flesh meat in any form should be avoided in the early stages. On the subsidence of the fever and the gradual diminution of the anasarca we may discontinue the more active measures, but the action of the skin should be carefully promoted. When convalescence has been fairly established, iron may be cautiously substituted for the alkaline and diaphoretic remedies. When tolerated, iron acts very beneficially and hastens in a marked manner the disappearance of blood and albumen from the urine; but it must be remembered that when begun too early, the preparations of iron are apt to induce a return of the acute symptoms. Hamburger[1] speaks strongly in favour of quinine in scarlatinal dropsy after the pyrexia has abated. He gives to children 1½ or 2 grains, and to adults 3 to 4 grains twice a day. Of forty-seven severe cases thus treated he obtained amendment in forty-four either immediately or in a few days. My experience leads me to conclude with Dr. Parkes that gallic acid exercises no beneficial influence in the acute disorder.

Patients continue liable to relapses for a considerable time, the slightest exposure being sometimes sufficient to induce the reappearance of the pyrexia, and of the albumen and blood in the urine. A complete suit of flannels is therefore essential, and, as a rule, the convalescent should not be allowed to leave his room until the albumen has disappeared from the urine. When this has taken place, or even before, if the case prove very lingering, change to a warm, sheltered locality is likely to prove highly beneficial.

The secondary thoracic inflammations present great difficulty in their management. They usually set in at a period when antiphlogistic measures cannot be borne, and they run their course with unusual severity and rapidity. Counter-irritants and revulsives may, however, be energetically employed, always avoiding cantharides and turpentine on account of their specific irritating effect on the kidneys. Dry-cupping over the chest is frequently of great service.

In the obstinate vomiting which sometimes prevails, creosote or small doses of chloroform in iced solutions may be given. The special treatment of uræmia will be considered in the article on chronic Bright's disease.

(B.)—CHRONIC BRIGHT'S DISEASE.

MORBID ANATOMY.—Three chief types of alteration are found in the kidneys of persons who have died of chronic Bright's disease, viz.: I. *Kidney smooth, white*, and enlarged; rarely in extreme cases atrophic (chronic nephritis). II. *Kidney granular*, brownish or red, and contracted (cirrhotic kidney). III. *Kidney lardaceous or waxy* (so-called amyloid degeneration). These types are not always found pure and simple, but, on the contrary, the main type is often complicated with superadded changes belonging to another type: thus the smooth white kidney becomes not unfrequently affected with waxy degeneration, while the granular and waxy forms are both liable to inflammatory attacks leading to changes belonging to the first type. The clinical history as well as the anatomical condition is thus rendered complex.

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[1] Prag. Vierteljahrschr. 1861.

I. *Smooth White Kidney.*

In this form the structural changes are essentially similar to those described as occurring in acute Bright's disease, but advanced to a further stage; the surface continues perfectly smooth, and presents conspicuous stellate patches of blood-vessels; the capsule is thin and easily stripped off; the organ is considerably enlarged, and on section the cortical substance is seen to be greatly increased, and to have an ivory-white appearance; or, if fatty, it is yellowish. The cones retain their usual colour, but appear conspicuously red from contrast with the abnormally white cortex.

The microscopic changes are essentially limited to the uriniferous tubes, which are distended and enlarged from the enormous increase of their epithelial lining; they sometimes contain transparent fibrinous exudation and blood. The cells are swollen, generally opaque and granular, and often loaded with oil. As the disease progresses, many of the tubes and their contents are broken up into a granular debris and absorbed. The intertubular stroma is, in typical examples, unaffected. The Malpighian corpuscles are but slightly, if at all, enlarged: their capsule is thin as in the natural kidney. The cones undergo changes analogous to those in the cortex, but less developed; fibrinous casts sometimes occupy the interior of the straight tubes.

The large smooth kidney generally remains large and smooth to the last; but in some cases, if the patient survive for some years, a progressive dwindling takes place, so that in extreme cases the kidney may not weigh more than an ounce, if so much. When atrophy has taken place, the capsule is somewhat thickened and adherent, and slight depressions are seen on the renal surface, giving the organ a slightly granular character. This atrophic condition, which Dr. Dickinson states is usually due to the superaddition of amyloid changes, seems to be produced by a destruction and gradual absorption of the distended tubules and their epithelial contents, leading to progressive disappearance of the cortex while the pyramidal portions remain intact. The blood-vessels are much thickened, and, according to Dr. Grainger Stewart, there is a relative increase of the fibrous stroma—much less, however, than in the cirrhotic kidney.

The large white kidney is not unfrequently the subject of fatty change, especially when the disease has arisen from cold; it constitutes one form of the "fatty kidney," and though this change may begin at an early stage of the disease, it only reaches an extreme degree in long-standing cases. When this condition is present, oily particles are found in great numbers in the epithelial elements, and lying free in the tubules.

SYNOPSIS OF SYMPTOMS AND CONDITIONS OF ORIGIN.—The smooth kidney is found in those cases in which chronic Bright's disease has followed on the acute disorder. The invasion of the disease has been sudden, and it can usually be traced to some definite exciting cause, either cold or scarlatina. I have also found the large white kidney in chronic Bright's disease following repeated pregnancies, and in a case arising in the course of phthisis.

The average age of 106 cases of smooth large kidney examined by Dickinson was 28·2 years; in eleven cases of smooth dwindled kidney it was 43·6 years; while in 250 cases of granular kidney it was 50·2 years.

The urine is generally scanty, of normal or slightly above the normal specific gravity, pale and cloudy—sometimes smoky and tinged with blood. On standing, it deposits a quantity of amorphous renal debris and tube-casts of various character—"epithelial," "fatty," "granular," and "hyaline." Cells resembling pus-corpuscles are common towards the later periods. Dropsy is an almost invariable coincident: the face is pale and puffy, and the cutaneous surface conspicuously white, smooth, and glossy. There is a markedly greater tendency to secondary inflammations and to uræmic accidents, but less to valvular heart disease and hypertrophy of the left ventricle, than in granular kidney.

The disease is of shorter duration than in granular kidney; in fatal cases the usual duration is under six months. But exceptional cases occur in which its course extends over several years. In these protracted cases the albuminuria continues long after the disappearance of the dropsy. I have known cases in which abundant albuminuria has persisted for more than a year after all other symptoms of disease had ceased; at length the albuminuria has gradually disappeared, and health been re-established.

Permanent recovery is not impossible after the lapse of a year or more.

II. *Granular Contracting Kidney.*

SYNONYMS.—Granular Kidney; Cirrhotic Kidney; Gouty Kidney; Intertubular Nephritis; Interstitial Nephritis.

The organ is diminished in size and reduced in weight—in extreme cases to two or three ounces or less. Its surface is rough and studded with numerous rounded elevations, varying from the size of a pin's head to that of a small pea. The capsule is opaque, thickened, and adherent to the subjacent surface, so that it cannot be stripped off without tearing the glandular structure; in places it dips into the cortical substance and divides the kidney irregularly, giving it a lobular appearance. On section, the cortex is manifestly atrophied, as compared with the cones, and forms around the bases of the pyramids a thin rim, only a line or even less in thickness; it has a red or brownish-red colour, and a coarse granular texture. The entire organ is tough and resistant. In the rare cases in which the granular kidney is met with in its early stage, thickening of the capsule and slight granulation of the surface are found to precede contraction, so that at this period the organ preserves its normal volume. In the contracted stage, the granular kidney allows injections to penetrate imperfectly. This diminished permeability is not entirely due to the cirrhotic state of the intertubular matrix, but partly, as Johnson has pointed out, to thickening of the walls of the minute arteries of the kidney.

On microscopic examination, the secreting tissue is found to have undergone extensive destruction. The Malpighian bodies are shrunk to half their size and abnormally crowded, their vascular tufts being embraced in a fibrous and granular investment, and, in extreme instances, compressed into an impermeable knot at the bottom of their capsules. Some of the uriniferous tubes are denuded of epithelium and reduced to mere tubular threads; some, also denuded, contain glassy fibrinous cylinders, while others are packed with broken-up epithelium. Both the fibrinous exudation and the disintegrated epithelium sometimes contain oil, but not so commonly nor so abundantly as in the smooth kidney. Comparatively normal tubes lined with healthy epithelium may be found side by side with tubes thus altered. A large quantity of adventitious connective tissue lies between the wasted structures, giving the organ its peculiar toughness.

According to Dr. Johnson, the morbid process begins in the epithelial cells, but Dickinson and Grainger Stewart describe it as originating in the intertubular matrix, and as consisting essentially in an enormous hypertrophy of the fibrous stroma of the organ. By the pressure and contraction of this fibrous material, the tubes and Malpighian corpuscles are extensively destroyed, and the size of the kidney is progressively reduced. According to Dickinson, the fibrous growth begins beneath the capsule and then penetrates into the interior of the cortex—the points where these fibrous processes penetrate are depressed, and if these are numerous and distributed with tolerable regularity, an appearance of superficial granulation is produced. The disease thus travels from the surface, and eventually involves the pyramids. Cysts are very commonly met with in the granular kidney. They vary in size from a pin's head to a hazel-nut, but many are so minute that they can only be detected by the microscope. Mr. Simon believes that they are formed by an immense dilatation of epithelial cells. The generally received and much more probable view, however, is that they are produced by obstruction of the uriniferous tubes with exudation, at intervals, or by compression of their walls at interrupted spots by the contracting adventitious tissue. The spaces thus enclosed become distended with a serous fluid, and are sometimes found lined with an epithelial layer. Their contents are not urinous, but consist of an albuminous saline solution. They occur both in the cortex and in the cones: in the cones they are sometimes elongated and placed end to end like a string of sausages (Dickinson).

SYNOPSIS OF SYMPTOMS AND CONDITIONS OF ORIGIN.—The granular kidney is found in the majority of those cases of Bright's disease which are chronic from the beginning—which commence insidiously and without definite exciting cause. The disease may run a latent course for months or years. The subjects of it are more advanced in years than those of the smooth large kidney. The urine is copious —three or four pints a day—and of low density; towards the termination of the disease, however, it becomes scanty, or even suppressed. The quantity of albumen is comparatively small; in rare cases it may be temporarily absent. There may be no deposit in the urine, or it may be so scanty as to escape detection; when present, it is slight, composed of hyaline and granular casts, with very slight admixture of epithelium, and rarely fatty. As a rule blood is absent.

In a quarter to half the cases there is no dropsy; when present it is commonly slight and limited to œdema of the ankles and legs, or puffiness under the eyes: it may disappear for a time, and then return. The cutaneous surface, though pale and anæmic, has not the conspicuous whiteness noticed in the preceding type; the features are often pinched and sallow. Cardiac hypertrophy is a very frequent concomitant, and a rooted constitutional cachexy is very often present.

The common predisposing causes are habitual intemperance, gout, lead poisoning, repeated exposure to cold, and extensively distributed atheromatous degeneration of the tissues.

As the two types which have just been described comprise the vast majority of cases of Bright's disease, much discussion has taken place as to whether Bright's disease is a single or a multiform affection—whether the smooth large white kidney merges into the granular red contracting kidney, or whether they remain distinct affections throughout. Reinhardt, Frerichs, Rosenstein, and German observers generally, take the former view, while the latter has been placed beyond reasonable doubt by the labours of Johnson, Wilks, and Dickinson in this country.* It is of course admitted that the large white kidney may become atrophic, and Johnson, Dickinson, and Grainger Stewart adduce several examples of such a change, but they insist that in its furthest stage of contraction the smooth dwindled kidney is still distinguishable from the granular red kidney.

Of twenty-six fatal cases of enlarged white kidney observed by Johnson, there was dropsy in twenty-four, or 92 per cent.; whereas, in thirty-three fatal cases of contracted kidney, dropsy was present only in fourteen, or 42 per cent.; hence he pertinently observes, "It is difficult to understand how it can happen that the majority of those patients who have reached the final stage of renal degeneration should escape the dropsy, which, in a greater or less degree, troubles nearly all those who die in what is assumed to be an earlier stage of the same disease."[1] Again, convulsions and secondary inflammations, as pneumonia and peritonitis, are more frequent with the smooth kidney; whereas, hypertrophy of the left ventricle, atheroma, and apoplexy are more frequent with the granular kidney.

III. *Lardaceous or Waxy Kidney.*

SYNONYM.—Amyloid Degeneration.

The organ is usually enlarged, sometimes diminished, smooth, or slightly roughened, and markedly tough and hard; the capsule peels off readily. On section, the cortex is bloodless, of a white or yellowish colour, with a smooth, waxy, translucent appearance, resembling bacon-rind; on the smooth cut surface little appearance of the natural secreting structure is seen, but it is dotted over with bright glancing points: these are the changed Malpighian bodies. The cones appear unnaturally red and distinct. This description applies only to extreme degrees; when slighter, the nature of the change can only be determined by means of the microscope or by applying a solution of iodine. When a thin section is highly magnified the waxy material is seen to affect mainly, sometimes exclusively, the blood-vessels. The deposit invades primarily the muscular coat of the arteries, which it renders abnormally transparent and thick, thereby diminishing the lumen of the vessel. The Malpighian corpuscles are earliest attacked; they appear as shining particles with thickened capsules; their vascular tufts are infiltrated with the waxy material. In advanced cases, the vasa afferentia with the arteries and capillary network of the cortex, and even the vessels of the pyramids, are similarly changed. The epithelial cells of the uriniferous tubes are commonly withered, often infiltrated with fatty molecules, but they do not appear to undergo the true lardaceous change. Hyaline-casts exist in some of the tubules. The stroma of the gland is much increased in quantity, but is not lardaceous.

The proper test for waxy degeneration is a watery solution of iodine, which imparts to the affected portions a deep mahogany brown colour, whereas the unaffected parts acquire a yellowish tinge only.

The liver and spleen are usually enlarged and lardaceous when the kidneys are so affected. Of seventy-seven cases collected by Rosenstein, the three organs together were affected in forty-eight, the spleen and kidneys in twenty, the liver and kidneys in four, and the kidneys alone in five cases.

The chemical nature of the waxy material has only recently been investigated. Virchow concluded that it belonged to the same group as starch and cellulose, because like them it yields a violet colour with iodine and sulphuric acid. But the analyses of C. Schmidt and Kekulé show that it contains nitrogen, and in almost exactly the same proportions as the protein compounds. It further resembles albuminous compounds in yielding a violet colour with the cupro-potassic solution, in dissolving completely in dilute caustic potash, and in being precipitated from this solution in white flocks by acids. The proportion per cent. of carbon, hydrogen, and nitrogen found by Kekulé in purified waxy matter from an exquisite specimen of lardaceous spleen was C. 53·58, H. 7·00, N. 15·4—which corresponds closely with the percentage of the same elements in albumen. Dickinson considers it a variety of fibrine, but differing from ordinary fibrine in containing about one-fourth less alkali and a somewhat larger proportion of earthy salts.

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[1] Med. Chir. Trans. vol. xlii. p. 156.

SYNOPSIS OF SYMPTOMS AND CONDITIONS OF ORIGIN.—Waxy degeneration of the kidneys always comes on insidiously and in cachectic persons debilitated by some pre-existing wasting disease. In 145 instances collected by Fehr, it coexisted with pulmonary tubercle in forty-three, with syphilis in thirty-four, and with caries and struma in twenty-six cases.[1] The course of the disease is essentially chronic.

The urine in the earlier stages is markedly copious—60, 100, and even 200 ounces per day. Dr. Grainger Stewart has pointed out the important fact, which my own experience fully confirms, that this polyuria is a marked feature, even before the urine becomes albuminous, and that this supplies a warning of the advent of this form of Bright's disease. As the disease advances the urine becomes scantier and of higher density, and the proportion of albumen, at first small, becomes very great. The colour of the urine is usually pale; there is a very scanty deposit which consists of casts and atrophied renal cells, which are sometimes fatty. Cells resembling pus-corpuscles are occasionally found either separate or aggregated round a cast. The tube-casts are usually hyaline, and do not yield a brown coloration with iodine: epithelial casts are also sometimes seen. Münch found corpora amylacea constantly present in the urine of a man with waxy kidney: they were coloured violet by iodine and sulphuric acid. Dropsy is present in the majority of cases; Fehr found it in 98 out of 152 cases collected by him: in some it is abundant and general, in others slight and partial. Uræmic symptoms are strikingly infrequent.

The diagnosis of waxy kidneys rests partly on the coincidence of a pale abundant albuminous urine with dropsy, but chiefly on the co-existence or pre-existence of one of the wasting diseases of which waxy kidneys are known to be a frequent complication, viz., phthisis, caries, long-continued suppuration, and constitutional syphilis.

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[1] A. Fehr, Ueber die Amyloide Degeneration, insbesondere der Nieren, Bern, 1867.

GENERAL COURSE AND SYMPTOMS OF CHRONIC BRIGHT'S DISEASE—In the great majority of instances chronic Bright's disease begins slowly and imperceptibly. The attention of the patient is awakened, some months, or it may be years, after it has existed, by the gradual failure of his strength and his increasing pallor or sallowness, with disinclination to exertion; or his suspicions are aroused by a little puffiness under the eyes, a slight swelling of the ankles at night, unusually frequent calls to void urine, or shortness of breath. In other cases these premonitions pass unheeded or are altogether wanting. The disease proceeds silently, amid apparent health, and then suddenly declares itself by a fit of convulsions, rapid coma, amaurosis, pulmonary œdema, or a violent inflammation; or it may lie concealed for an undetermined period, and then reveal itself, after exposure to cold or a fit of intoxication, in the guise of an acute attack, with rapid general anasarca and scanty sanguineous urine; or it may be a continuation or sequela of acute Bright's disease; or, lastly, it may creep on stealthily in the wake of some pre-existing chronic disorder—phthisis, caries, necrosis, constitutional syphilis, gout, chronic alcoholism, or exhausting suppuration.

The principal symptoms of the disease are:—albuminous urine with deposits of tube-casts and renal epithelium; frequent micturition, especially at night; dropsical effusions into the subcutaneous cellular tissue, serous cavities, or pulmonary substance; dryness of the skin; derangements of digestion; progressive hydræmia; uræmic phenomena (headache, amblyopia, convulsions, coma, vomiting, and diarrhœa); hypertrophy of the left ventricle; secondary inflammation of the parenchymatous organs and serous membranes.

Few cases present the whole of these symptoms; many present only two or three of them. The alterations in the composition of the urine are the most invariable, and also the earliest and most distinctive symptoms; next follow in the order of constancy, the deterioration of the blood, the dropsical symptoms, and, lastly, the uræmic and inflammatory incidents.

The disease usually pursues an interrupted course, being subject to occasional exacerbations, with intervals of quiescence. The exacerbations are generally induced by exposure to cold or some imprudence in diet or regimen; sometimes no cause can be assigned for their occurrence. They are marked by pyrexia, and often simulate an attack of acute Bright's disease. The intervals of quiescence may be some weeks or months, or a few years; the remission of the symptoms is commonly only partial, the main features of the disease persisting, though in a modified degree. Sometimes, however, the remission is almost complete, and little except the albuminous state of the urine remains to attest the existence of renal mischief; and even this may, in exceptional cases, be absent, and the nature of the case be first revealed at the autopsy. After each exacerbation it is commonly pretty evident that the disease has progressed a step, and that probably an additional portion of the kidney, hitherto spared or only slightly affected, has been disabled. The kidneys are at length so seriously disorganised, and their depurative functions so far abrogated, that life becomes impossible.

The immediate cause of death is variable. Sometimes the sufferer passes quietly away exhausted by anæmia, burdensome anasarca, and defective digestion. About one-third of the subjects of chronic Bright's disease perish by uræmic poisoning, either in the form of coma and convulsions, or irrepressible diarrhœa and vomiting. A considerable number die from the intensity or dangerous situation of the dropsical effusion, as when the glottis or lungs are invaded; or death results from hydrothorax, or from gangrenous erysipelas set up in the tense œdematous integuments of the legs, thighs, or genitals. About one-fifth die by secondary pneumonia, pericarditis, or double pleurisy. The remainder are cut off by more remote complications, as apoplexy, cirrhosis, phthisis, intestinal ulcerations, &c.

The *duration* of the disease can only be approximately ascertained from the difficulty of assigning the exact date of invasion. Enough is, however, known to show that it varies within very wide limits. The usual period is from two to three years; but cases may end in six months, or be protracted for four or five years. Exceptional instances have been recorded in which patients have survived ten years and even fifteen and twenty-three years.

PARTICULARS OF SYMPTOMS AND COMPLICATIONS.—*Urine.*—The quantity of albumen is most variable. The urine may become absolutely solid on boiling—or it may contain only the minutest traces of albumen, even in confirmed and fatally-tending cases. The amount of albumen lost in twenty-four hours varies commonly from 45 to 300 grains; Dr. Parkes observed in one instance 545 grains. During digestion the quantity is larger (it may be double) than during fasting; it rises and falls irregularly in the course of the disease, sometimes diminishing to a trace, and anon increasing to an intense impregnation.

The urine is generally pale and slightly turbid, depositing, on standing, an amorphous whitish sediment of renal epithelium and tube-casts. It sometimes contains blood—occasionally in quantity, but generally in microscopic proportion. When there is intercurrent pyrexia or the case is complicated with phthisis or regurgitant heart disease, the urine may be high-coloured and turbid from lithates.

The quantity of urine voided per diem varies with the type of the disease, and the presence or absence of pyrexia, sweating, vomiting, or diarrhœa. The specific gravity is low when the urine is copious (1,006 to 1,015); but when scanty, the specific gravity may rise to 1,030 or even 1,040. The urine is nearly always acid, and not unfrequently deposits uric acid and oxalate of lime; occasionally I have noted it alkaline from fixed alkali, and on two occasions ammoniacal on emission.

The *renal derivatives* (see Figs. 2, 3, 4) are markedly scantier in chronic than in acute Bright's disease; they are not unusually entirely absent for limited periods. They are, however, sometimes discoverable when the urine has temporarily ceased to be albuminous. The

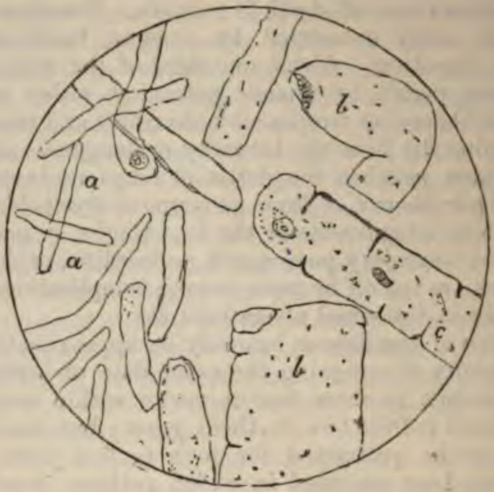


FIG. 2.—Transparent casts. *a a.* From the urine of a man with chronic Bright's disease of eight months' duration (urine bloody, intensely albuminous, anasarca, dying from pneumonia); *b b.* from a case of chronic Bright's disease (large white kidney); *c.* from a case of chronic Bright's disease (contracted kidney with fatty degeneration).

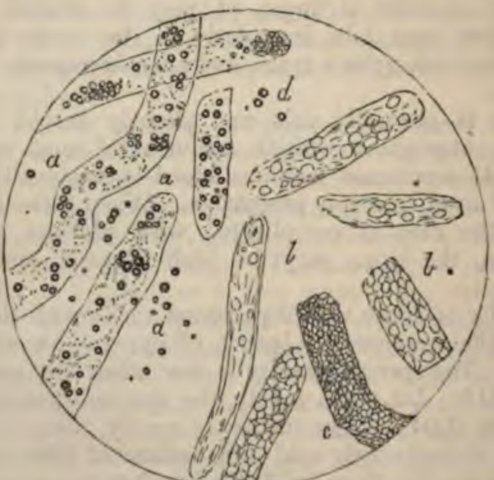
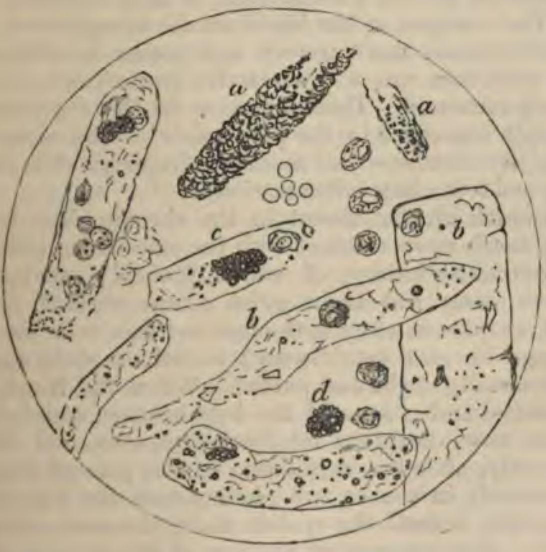


FIG. 3.—*a a.* Fatty casts; *b b.* blood-casts; *d d.* free fatty molecules.

thelial cells may be simply withered; more rarely they are totally ntegrated into an amorphous granular debris; in other cases they tain specks of oil, or they may even be wholly converted into an lomeration of oily particles, so as to appear identical with the ranular corpuscle" or "inflammation globule." If the casts are ilarly speckled with fat, and free oily dots are scattered over the d, it indicates a fatal disorganisation of the organs—either large y kidneys or contracted granular ones. Considerable diversity in character of the casts discharged by the same individual, even ing the same day, may be met with, arising from the different con- ion of the several parts of the gland: conclusions as to the probable te of the kidney can only be drawn from the *prevailing* character



G. 4.—Casts and renal cells from a case of contracting granular kidneys; *a a.* granular opaque casts; *b b.* hyaline casts; *c d.* fatty masses.

he deposit, and not from one or two individual casts or cells. The s *most commonly* seen in chronic Bright's disease are "small" and ge" hyaline forms and "granular" opaque ones, any of which may e a few wasted epithelial cells strewed over them. Perfect ithelial" casts are rare; blood casts are also rare, in chronic cases, ss there be concomitant tricuspid regurgitation. Large hyaline s result from the exudation having been thrown into tubuli uded of their epithelium; while if these have subsequently under- e contraction, the casts will be small and hyaline. Other tubuli, hed or partially clothed with epithelium, shed some of their cells the contained exudation, and cause the appearance in the urine of casts more or less studded with epithelial remnants. The longer the exudation is retained within the tubuli, the darker and more granular the casts; and *vice versâ*, casts speedily discharged are commonly hyaline. Sometimes casts are darkened by the colouring matter of the blood; and the opaque granular ones are sometimes composed of crushed epithelial debris moulded into the form of the tubuli.

The normal solids of the urine are all diminished in chronic Bright's disease. The urea is, as a rule, markedly reduced, the daily quantity averaging only about 100 grains; Frerichs has observed it as low as 15 grains. A case is mentioned by Mosler,[1] however, in which 640 grains were voided in one day! There is no correspondence, direct or inverse, between the urea-excretion and the discharge of albumen. With intercurrent pyrexia the excretion of urea increases.

Blood.—The changes in the blood are the complement of those in the urine; it becomes more watery and poorer in albumen and red corpuscles, while urea, uric acid, extractive matters and pale corpuscles, are relatively increased. This alteration in the composition of the blood is deeply concerned in the production of the more prominent features of the disease—the anæmia, dropsical effusions, uræmic phenomena and secondary inflammations.

Dropsy is much oftener absent in the chronic than in the acute form. It is much more constant with the smooth large than with the granular contracted kidney, of which probably one-third or one-fourth of the cases run their entire course without dropsy. The effusion begins quite as often in the feet and legs as in the face, and is apt to change its seat capriciously; sometimes it is excessive and general, but usually slight and partial. When the heart or liver is diseased, ascites and œdema of the legs become unduly prominent. The effusion may disappear totally for months, and then return; more frequently, after the subsidence of the general dropsy, œdema lingers obstinately in one or two places—over the flat of the tibiæ, about the ankles, beneath the eyelids, under the conjunctiva, or about the genitals. The presence or absence of dropsy generally, but by no means always, corresponds with the abundance or scantiness of the urine; but it has no relation to the amount of albumen.

The skin is usually obstinately dry; perspiration is quite exceptional, and is commonly due to diaphoretic treatment. Profuse sweating does, however, sometimes take place spontaneously, and may even continue for weeks; in one such case under my care, an abundant crop of pemphigus bullæ appeared. The integuments in some cases are excessively pale and glossy, more commonly sallow and rough. There is little or no tenderness in the renal region in the chronic cases, and the frequency of micturition is mostly observed at night. Some degree of bronchitis is almost an invariable coincident in both acute and chronic Bright's disease.

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[1] Archiv d. Vereins, Bd. xi. p. 513.

Complications and connection with other diseases.—The digestive organs are nearly always disturbed: at first there is anorexia and nausea, and later, frequent or even uncontrollable diarrhœa and vomiting are not unusual. In some cases these symptoms are obviously of uræmic origin; but not unfrequently anatomical lesions —follicular catarrh, dysenteric ulcers, sometimes with sloughing of the mucous membrane—are found in the intestines, which explain these disturbances. Treitz states that urea is discharged into the intestines from the blood, and being converted into carbonate of ammonia, acts as an irritant on the intestinal mucous membrane. In 220 cases of Bright's disease, collected by him, the following conditions of the intestines were found after death: Hydrorrhœa (intestines filled with yellow-greenish fluid) in 80; blenorrhœa and catarrh in 60; croupous and ulcerous dysentery in 19; sloughing in 12; sanguineous contents without discoverable source of hæmorrhage in 4; normal fæces in 5; and contents of intestines undetermined in 11 cases.[1]

Secondary inflammations of the lungs, endocardium, pericardium, pleura, peritoneum, or integuments may break out at any period in the course of Bright's disease, and the tendency to these constitutes one of the principal dangers of the complaint.

Cardiac hypertrophy, valvular disease, and pulmonary tubercle are frequent complications.[2]

Bright's Disease and Phthisis.—This complication is of frequent occurrence. In the great majority of cases the pulmonary disease is far advanced before renal symptoms appear, the long-continued discharge of pus from the lungs at length giving rise to waxy changes in the kidneys, followed by albuminuria and dropsical effusion. But sometimes the renal disease precedes the pulmonary, and the changes found in the kidneys after death are not invariably of the waxy type.

The co-existence of two fatally-ending diseases might have been expected to accelerate the inevitable issue; yet most of these cases run an exceedingly chronic course, continuing *in statu quo* for months together. In a case recorded by the writer, the pulmonary disease already in its third stage completely retrograded, and was supplanted by the renal affection.[3]

Bright's Disease and Diseases of the Heart and Vascular System.—The connection of cardiac disease with renal disorder is at least threefold.

1. *Simple hypertrophy of the heart,* especially of the left ventricle, is found without valvular incompetency and without degeneration of the muscular fibres. In this numerous class the cardiac is secondary to the renal affection. Bright, who was the first to point out this curious

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[1] Prag. Vierteljahrschr. 1859.

[2] For a table showing the proportionate frequency in 406 autopsies, with which the various organs, other than the kidneys, were found affected in Bright's disease generally, the reader is referred to the writer's work on Urinary and Renal Diseases, 3rd Edit. p. 413.

[3] Ibid. p. 414.

coincidence,[1] offered two explanations of it—that the altered composition of the blood either exercised an irregular and unwonted stimulation upon the muscular tissue of the heart, or so impeded the circulation in the capillaries that a greater effort of the ventricle was required to propel the blood through them. Traube believes it to be a conservative or compensating change, similar in its production to that induced by valvular incompetency or aortic constriction. He brings forward evidence to show that hypertrophy of the left ventricle is an almost constant concomitant of granular, contracted kidney. The destruction of secreting structure in these organs leads to the diminution of the amount of blood passing from the arterial into the venous system, and of the quantity of fluid withdrawn from the arterial system for the formation of urine. Both circumstances, but especially the second, operate to increase the tension in the arterial system, and consequently to increase the resistance which the left ventricle has to overcome in discharging its contents. If the compensation be complete, the increased tension in the arterial system occasions a larger transudation of water, and even of urea and other urinary solids, through the kidneys, and so materially aids in warding off dropsical effusion and uræmic symptoms. But, as in some apposite examples adduced by Traube, should some additional obstruction to the circulation arise through intercurrent inflammation of the bronchial tubes or of the lungs, pleura, or pericardium, the heart, enlarged and strengthened though it be, no longer suffices to overcome the increased resistance, and uræmia or dropsical effusions speedily manifest themselves.[2]

Renewed interest in the connection between chronic Bright's disease and cardio-vascular changes has been awakened by recent researches. Dr. George Johnson discovered that there is a widespread hypertrophy of the muscular walls of the small arteries in chronic Bright's disease. He finds it not only in the arteries of the kidneys, but also in those of the pia mater, the skin, the intestines, and the muscles. He explains the production of this hypertrophy, and the manner in which it reacts on the left ventricle, as follows:—"In consequence of the degeneration of the kidneys the blood is morbidly changed. It contains urinary excreta, and it is deficient in some of its own normal constituents. It is, therefore, more or less unsuited to nourish the tissues—more or less noxious to them. The minute arteries throughout the body resist the passage of this abnormal blood. The result of this antagonism of forces is, that the muscular walls of the arteries, and those of the ventricle of the heart, become simultaneously and in an equal degree hypertrophied. The persistent overaction of the muscular tissue, both cardiac and arterial, is found registered after death in a conspicuous and unmistakable hypertrophy."[3]

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[1] Guy's Hosp. Reports, vol. i. p. 396.

[2] Ueber den Zusammenhang von Herz und Nieren-krankheiten, p. 58; and Deutsche Klinik, 1859, p. 315.

[3] Lectures on Bright's Disease, p. 67.

Sir William Gull and Dr. Sutton have brought forward a novel view of the pathology of granular kidney and of the associated changes in the cardio-vascular system. They believe that there exists in these cases a peculiar pathological change to which they give the name of "Arterio-capillary fibrosis." This consists in the deposit of a "hyalin-fibroid" material in the fibrous coats of the arterioles and capillaries. This change may prevail extensively throughout the vascular system, in the kidneys, skin, pia mater, heart, lungs, spleen, stomach, and retina. In its nature it is allied to, but not identical with, senile changes. It commonly begins in the kidneys, but it may begin elsewhere; so that hypertrophy of the heart, with degeneration of the arterioles, may be found associated with healthy kidneys; and that when atrophy with granulation of the kidneys exists, it is but part and parcel of a general morbid change. The granular form of Bright's disease is, in their eyes, essentially an instance of arterio-capillary fibrosis. These views have been sharply criticised, and are still in dispute, but they have the merit of bringing into true prominence what every observer of extended experience in cases of granular kidneys must have had strongly impressed on his mind—namely, that the renal affection is not a mere local affair, but that it is, rather, a part of a wide-spread tissue-degeneration involving the entire or a large portion of the body.

2. *Valvular defects* and their consequences frequently co-exist with Bright's disease. Some of these are examples of endocarditis, secondary to the renal disease; but in other cases, the cardiac and renal affections arise independently of each other; or they depend on some common cause, as in a case recorded by myself,[1] in which fatty degeneration had simultaneously invaded the heart, brain, and kidneys, giving rise to three distinct groups of symptoms.

3. In a third class are comprised all those cases in which the renal disorder is secondary and subordinate to cardiac disease. They are described in the article on Congestion of the Kidneys, to which the reader is referred.

URÆMIA.

The phenomena to which the term *uræmic* has been applied consist of twitchings and convulsions of the voluntary muscles, headache, drowsiness, coma, defects of sight and hearing, vomiting, and diarrhœa.

It is noteworthy that uræmic phenomena of a paralytic nature affect the sensorium and the special senses, but not the voluntary muscles; while those of an opposite kind (exalted irritability) affect the voluntary muscles, but not the sensorium. Delirium is rare while coma is frequent; paralysis of the limbs is scarcely known unless there be some anatomical lesion of the brain superadded, while convulsions are frequent.

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[1] Op. cit. p. 421.

The mode in which uræmic symptoms appear and the forms they assume are very various. They generally begin insidiously with headache and vomiting, followed by heaviness, indifference, and somnolence. These premonitory symptoms may pass off in a few days or they may be succeeded by general convulsions and coma. In other cases the patient is struck down with convulsions or insensibility without any previous warning, or he becomes suddenly blind, or is seized with uncontrollable vomiting.

Headache is the most common uræmic symptom; it is seldom continuously absent in degeneration of the kidneys. A sense of heavy weight or compression is complained of over the forehead or vertex, or there is obstinate pain at the back of the neck or behind the orbits.

Defects of Sight.—These consist either in amblyopia—a dimness of vision which comes and goes, objects appearing as if veiled in mist—or in rapid and complete, though usually transient, blindness. Temporary loss of sight often accompanies the convulsive seizures and usually persists for a time after they have passed off. The ophthalmoscope reveals no organic change in the eye in genuine uræmic amblyopia; it is purely cerebral, and not to be confounded with the hæmorrhagic blindness (retinitis apoplectica), which is also common in Bright's disease, and in which the loss of sight, though seldom complete, is more permanent. The latter affection is due, as Von Gräfe has shown, to rupture of the retinal vessels, and is akin to the sanguineous apoplexy to which sufferers from Bright's disease are liable: its production is probably due to the hypertrophy of the left ventricle, which so commonly accompanies a contracting kidney, and the increased arterial tension consequent thereupon.

Uræmic deafness is much less common than amblyopia; its occurrence is highly exceptional.

Uræmic convulsions conform closely to the epileptic type as a rule; they usually leave the patient comatose. In exceptional cases consciousness is not wholly lost. In a lady under my care the paroxysms coincided with the catamenial periods; during the convulsions the patient knew the persons about her, and called loudly to be held fast. Dr. Bright relates a case in which intelligence remained perfect, although the patient was in a state of convulsion, with forcible drawing up of the legs and distortion of the muscles of the face.

An attack of uræmic convulsions may consist of a single paroxysm, or, more frequently, of a succession of such paroxysms, following each other at uncertain intervals of a few minutes or hours, the patient during the remissions lying in a state of profound insensibility, with stertorous breathing, pale face, and dilated pupils; or in deep drowsiness, but capable of being partially roused when spoken to or shaken. If a first attack does not prove fatal, it may recur at irregular intervals of weeks or months, or be replaced by other uræmic symptoms.

Uræmic coma either comes on insidiously, passing into complete stupor in the course of two or three days; or the patient falls down as if in apoplexy, perhaps while walking in the street, or pursuing his usual avocation. When there is no anasarca, and the previous state of the urine is unknown, cases of this class are very liable to be confounded with apoplexy or with narcotic poisoning.

The *diagnosis* of uræmic coma from apoplexy rests on the absence, in the former, of paralysis and the partial recovery of consciousness between the convulsive attacks, if there be any. From ordinary epilepsy, the diagnosis is sometimes difficult, if there be no known antecedent history to indicate the nature of the case. The incidents of the seizures are often identical, even to the existence of an aura; but as a rule, the turgid purplish countenance and asphyxial character of true epilepsy are absent in uræmic fits, in which the face is nearly always deadly pale and the breathing easy. From opium-poisoning, renal coma is distinguished by the dilated or semi-dilated pupils, and by the occurrence of remissions in the insensibility. Dr. Richardson relates the cases of two children poisoned by belladonna berries, in which the symptoms closely resembled uræmic coma sequential to scarlatina; the insensibility was complete, and the pupils strongly dilated. The examination of the vomited matters and of the urine furnishes the best means of diagnosis in such cases.

In all cases of convulsions or insensibility from doubtful causes, the urine should be examined, and, if necessary, withdrawn by catheter for the purpose. It ought to be remembered that sanguineous apoplexy is not very unfrequent in chronic Bright's disease.

Uræmic coma and convulsions may occur separately; but much more commonly the attacks are of a mixed character, and combine several or all the phenomena just enumerated.

As a rule, the quantity of urine and the excretion of urea diminish notably immediately before a uræmic attack; but sometimes great scantiness of urine, or even, in acute Bright's disease, total suppression may exist without uræmic symptoms. Biermer relates a case of scarlatinal dropsy in which complete suppression of urine continued for five days without uræmia; for four days and a half afterwards a few teaspoonfuls a day were secreted, and yet no uræmia; after this the urine flowed abundantly for a short time, and then again became scanty: three days later uræmic coma set in, followed by convulsions, which proved fatal.

Uræmic vomiting and diarrhœa are common phenomena of Bright's disease, but the vomiting is not always uræmic, there being greatly impaired digestive powers throughout the disease. Genuine uræmic vomiting takes place without reference to the contents of the stomach, and is frequent or uncontrollable, the vomited matter being a watery fluid, either distinctly ammoniacal to the smell, or, if acid, evolving ammonia freely when caustic potash is added. The alvine dejections in uræmia are similarly characterized.

Paroxysms of dyspnœa belong to the least frequent forms of uræmic disturbance, if indeed such attacks have at any time a genuine claim to the designation uræmic. Fournier cites some cases of this kind; and one somewhat doubtful example has come under the observation of the writer.

Theories of Uræmia.—As morbid anatomy throws no light on the determining cause of uræmia, observers have been led to assume that that cause consists in the retention in the blood of some excrementitious material (or its derivatives), which is normally removed out of the body by the kidneys; but which being retained renders the blood incapable of ministering to the normal operations of the nervo-muscular system. Hence the various abnormities of motion and sense which have just been described. Hammond and Richardson, following the original notion of Willis, contend that the special poison in these cases is urea. Frerichs, on the other hand, maintains that urea is itself innocuous; that uræmic symptoms depend upon the transformation of the urea in the blood into carbonate of ammonia. He supports this view by showing that carbonate of ammonia invariably exists in the blood of uræmic patients, and that if injected into the veins it produces fits of convulsions with intervening periods of coma exactly resembling genuine uræmic attacks. Treitz likewise believes that carbonate of ammonia is the poisonous agent, but that urea is first vicariously excreted into the alimentary canal, where it is speedily converted into carbonate of ammonia and then absorbed into the blood. This theory furnishes at least a rational explanation of uræmic vomiting and diarrhœa, and of the presence of the volatile alkali in the excreta. That urea is excreted by the intestines in Bright's disease is undoubted, and its rapid conversion into carbonate of ammonia has been proved experimentally by Bernard. Richardson and Hammond however have, since the promulgation of Frerichs' theory, found that ammonia naturally exists in the blood of healthy animals; and all subsequent observers, Petroff of Dorpat alone excepted, have failed to discover a larger amount of ammonia in the blood of animals rendered uræmic by the removal of their kidneys, than exists in the healthy state. It has also been shown that urine, urea, and chloride of sodium, as well as carbonate of ammonia, are capable of producing uræmic symptoms when injected into the blood.

The urea and ammonia theories of uræmia seem to be set aside by the recent experiments of Oppler, Schottin, Perls, and Zalesky, which indicate that uræmic phenomena depend mainly and essentially on the accumulation in the blood and tissues of those primary products of tissue-metamorphosis, creatine, creatinine, and other extractives, which in a later stage of histolysis are converted into urea and uric acid. These experiments tend to show that it is not improbable that urea and uric acid are actually formed *by the kidneys*, and that any traces of them found in the blood are due to re-absorption from the urinary channels.

Inquirers into the theory of uræmia may be reminded of the remarkable absence, in cases of obstructive suppression of urine, of coma and convulsions, the most common clinical features of uræmic intoxication.[1]

Dr. Owen Rees believes that the tenuity of the blood in Bright's disease is not without influence in the production of the cerebral symptoms. Traube has still further developed this idea. He contends that the watery state of the blood predisposes to interstitial transudations, and that when from any cause the tenuity of the blood-serum is still further increased, serous transudation takes place through the cerebral capillaries, giving rise to œdema of the brain. This œdema compresses the minute cerebral vessels, producing cerebral anæmia and thereby uræmic coma and convulsions; when the hemispheres are affected, he believes coma is produced; and when the central ganglia are involved, convulsive phenomena are the result.

After a careful review of the observations and experiments adduced on all hands, I have to express my conviction that none of the exclusive theories of uræmia have made good their claim to acceptance. The subjects of Bright's disease suffer under a vitiated condition of the blood and tissues: the blood is unnaturally watery and deficient in albumen; the blood and tissues are impregnated with the primary histolytic products, such as creatine, extractives, &c., and with excrementitious urinary compounds (urea and uric acid), perhaps also with some of their products of decomposition. This state appears to induce in the nervous centres a proneness to sudden disorder and loss of equilibrium, which may be developed at any moment by an exaltation of one or several of the disturbing elements, or by the supervention of some new and different source of irritation. This state of erethism is analogous to that which naturally prevails in children: in whom an irritation which would be of no moment in an adult suffices to awaken convulsive and comatose phenomena closely resembling those of uræmia.

DIAGNOSIS OF CHRONIC BRIGHT'S DISEASE.—Under ordinary circumstances, the symptoms and the condition of urine are so characteristic, that the disease can scarcely be confounded with any other: a persistently albuminous state of the urine, apart from heart disease, even in the absence of dropsy, hardly belongs to any other condition.

Cases of temporary albuminuria in febrile complaints, without structural changes of any importance in the kidneys, differ from Bright's disease in the absence of dropsical effusion; the quantity of albumen is also generally very small, and totally disappears on defervescence; the urea-excretion is natural, or even excessive, instead of being diminished.

The real diagnostic difficulties lie:—(*a*) in distinguishing acute and curable cases from chronic confirmed ones; (*b*) in determining the precise anatomical changes going on in the kidneys; and (*c*) in detecting the disease when masked by an inflammatory complication or a uræmic paroxysm.

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[1] See Urinary and Renal Diseases, 3rd Edit. p. 29.

(*a*) The case must be considered as belonging to the chronic and confirmed class if the disease has crept on insidiously, or if it is complicated with chronic phthisis, caries, long-continued suppurations, constitutional syphilis, enlarged liver or spleen, or hypertrophy of the left ventricle.

If the invasion has been acute, and albuminuria still lingers after the febrile symptoms have abated, time is required to establish the diagnosis. The fear that the disease has become confirmed grows stronger every day that passes without diminution of albumen in the urine. The characters of the urinary deposit supply important information at such a juncture. If the epithelial elements and blood-corpuscles are freely discharged, and the renal derivatives show little or no sign of fatty change, the probability is that we have to deal with the declining stages of an acute disorder. If, on the other hand, albumen persists in considerable quantity after the acute symptoms have passed away, and after blood has almost or quite ceased to appearin the urine, the disease has probably lapsed into a chronic and confirmed state; if, besides these untoward signs, the deposit shows marked fatty change, that probability becomes a certainty.

It must not be forgotten that occasional febrile exacerbations occur in chronic Bright's disease, in which the urine becomes scanty, high-coloured, and perhaps bloody. These are liable to be confounded with the acute disorder, and when there are no clear indications of chronicity in the previous history, in the character of the renal derivatives, or in the co-existence of complications, the differential diagnosis may be quite impracticable until the lapse of time shall have cleared up the ambiguity.

(*b*) For the differential diagnosis of the types of degeneration going on in the kidneys the reader is referred to the synopsis of distinctive symptoms appended to the description of each anatomical type.

(*c*) When the case comes under observation masked by an inflammatory complication, such as pneumonia, or endo- or pericarditis, a clue to the primary affection must be sought in the previous history of the case, and in the associated symptoms. The primary renal disease is apt to be overlooked, and the case regarded as one of simple inflammation of the organ affected, if dropsy is absent, and there is no history of any. In such cases the urine assumes a febrile character, urea becomes abundant, and its specific gravity rules high; if, under such circumstances, the quantity of albumen in the urine be but small, the absence of Bright's disease may be counted on. The converse deduction is not, however, invariably warranted; in pneumonia I have seen the urine for some days "highly" albuminous, as the sequel showed, without the existence of any renal degeneration.

In pneumonia and in pleurisy, when the urine is albuminous, the simultaneous implication of both sides furnishes a strong presumption that the inflammation is not simple but secondary to renal disease. The existence of notable anæmia, or of cardiac hypertrophy, without valvular disease, also favours the supposition of Bright's disease. The differential diagnosis of uræmic coma and convulsions has been pointed out under uræmia.

The absence of casts, generally more apparent than real, in albuminous urine, gives no security against the existence of renal degeneration. When the casts are small and few in number they subside very imperfectly, and are apt to escape detection even with the most careful examination. In other cases the absence of casts is only temporary; and I have known it most absolute in some of those hopeless cases where the renal disease is the ultimate issue of an inveterate strumous or syphilitic cachexia.

PROGNOSIS OF CHRONIC BRIGHT'S DISEASE.—The prognosis in confirmed chronic Bright's disease is exceedingly gloomy. The textural changes in the kidneys are of a kind that do not admit of reparation. The Malpighian bodies become enveloped in an exudation of low plastic material, tending to progressive contraction, and the tubuli are either blocked up with fibrinous plugs, or shrivelled into useless fibres. The disease advances unequally in different parts of the gland, and the sounder portions carry on, in an increasingly imperfect manner, the depurative functions, until the blood becomes so loaded with histolytic and urinous elements as to be incompatible with life. Long before this extreme limit is reached, however, death is caused in a large number of cases by one or other of the numerous complications to which the subjects of renal degeneration are liable.

But although the final prognosis in chronic and confirmed cases is thus unfavourable, in certain cases the structural changes cease to advance, the dropsical effusions, if any existed, are absorbed, and the condition of the patient remains stationary for months, or perhaps for years; nay, even in the more unfavourable cases there is still hope that by judicious management amelioration of the more distressing symptoms may be brought about. Except in the ultimate stages of the disease, the dyspeptic symptoms, the irregularities of the bowels, the dropsical accumulations, and the bronchial catarrh, may be combated with good probability of success. Cases protracted to five or six years are not uncommon, and a few instances are recorded in which the patient has survived for ten, fifteen, or even twenty years; in some rare cases the disease does not prove fatal at all. Possibly in these cases only one kidney is affected, its fellow remaining sound; this may be inferred, not only from the great difference in the amount of disease sometimes found in the two kidneys after death, but with greater certainty from such a case as that recorded by Dr. Moxon, in which one kidney was in an advanced stage of degeneration while its fellow was normal.[1] The tenure of life under these circumstances is exceedingly precarious; and an imprudent indulgence or exposure may bring life, in a few hours or days, to the verge of destruction.

The favourable and unfavourable signs in Bright's disease have relation to the state of the skin, the duration of the disease, the degree of deviation of the urine from its natural quantity and composition, and the existence of complications.

The signs which indicate that an unfavourable termination is not far distant are:—obstinate dryness of the skin, steady decrease of the urine, which had been previously abundant without proportionate increase in its density—evidence that the disease has existed some years, repeated recurrence of uræmic phenomena, excessive serous effusion, excessive cardiac hypertrophy, a persistently restless state. Speedy death is indicated by the breaking forth of pneumonia or pericarditis, by suppression of urine, or uncontrollable vomiting and diarrhœa. The absence of these signs may be construed in a favourable sense as indicating a stationary condition and the probability that the final issue may be yet far distant.

An excessive proportion of albumen in the urine, although a proof of the activity of the morbid process, and therefore a sign of evil import, is not necessarily prophetic of impending death. In a case observed by me, the urine, which was examined almost daily, became constantly solid on boiling, for a period of more than two months; during this time the patient's condition remained stationary; he was then seized with pneumonia, of which he speedily perished.

TREATMENT OF CHRONIC BRIGHT'S DISEASE.—In the management of cases of confirmed Bright's disease, three objects are to be especially aimed at, namely: (*a*) to hinder the further extension of the structural changes in the kidneys; (*b*) to prevent the occurrence of uræmic and inflammatory accidents; and (*c*) to palliate or remove certain threatening or troublesome symptoms, such as anæmia, dropsy, and dyspepsia.

To fulfil the first indication, the conditions under which the complaint originated must be carefully traced out, and the patient removed as completely as possible from their further influence. In some instances this is practicable: as when the disease follows intemperance or long-continued exposure to wet and cold. In protracted suppurations, necrosis, caries, joint-disease, stricture of the urethra, and old vesical inflammation, the possibility of the development of renal degeneration should be kept in view by the surgeon, and should have weight in considering the propriety of operation. In all such affections the condition of the urine should be narrowly watched, the first appearance of albumen being a warning that the opportunity for operative procedures is passing away, never to return.

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[1] Path. Soc. Trans. vol. xix. p. 268.

There is no evidence that local counter-irritants of the severer class—issues, setons, and the like—applied over the kidneys, exert any good effect; and the ulcerations which they sometimes leave are apt to prove intractable. Mustard-poultices, tincture of iodine, and dry-cupping, may be applied when the loins are the seat of aching pain, but their influence on the renal lesion is probably *nil.* Blisters are inadmissible on account of their specific irritating effects on the urinary system.

The patient should be completely clothed in flannel, and the activity of the skin should be promoted by moderate walking or carriage exercise, and the occasional use of warm baths and frictions of the surface. The bowels should be opened at least once daily, and the diet should be light and nutritious. Milk agrees well as a rule, and may be freely taken. Two or three glasses of claret or hock daily, or a glass of sound beer, are permissible; but the stronger wines and all spirits usually agree badly, and should not be allowed except when special circumstances imperatively call for their administration.

Saturation of the system with iron is the best safeguard against the profound anæmia accompanying chronic renal degeneration. I have been in the habit, when the secondary symptoms or complications do not call for special treatment, of giving fifteen or twenty-five drops of the muriated tincture of iron in a wine-glass of water night and morning; combined, in cases with strumous tendency, with cod-liver oil.

If the tincture produces headache, or disturbs digestion, the citrate of iron, with or without quinine, the syrup of the phosphate or of the iodide of iron, the saccharated carbonate, or the ferrum redactum, may be given. Certain substances, such as the mineral acids, especially nitric acid, iodide of potassium, tannin and gallic acid, are said to be capable of diminishing the quantity of albumen lost by the urine. Dr. Parkes exhibited large doses of tannin and gallic acid without producing any diminution of albumen; and I have in a number of cases used gallic acid for many weeks without any favourable influence on the excretion of albumen; in some instances it occasioned serious gastric disturbance. Oppolzer has recommended alum—the trials of which by Heller seem to have been satisfactory.

When a stationary condition is maintained the treatment should be limited to the enforcement of sound hygienic rules and preventive measures. The patient should be made clearly to understand that he is to treat himself as a valetudinarian; and that in his clothing, his eating, drinking, exercise, and general mode of life, he must go by rule if he would avoid running the most fatal risks.

The *dropsical effusions* are most effectually combated by hydragogue cathartics and warm baths. For general use there is no hydragogue superior to the compound jalap powder; it acts quickly, and produces two or three copious watery stools. The nausea and sickness, which it too often occasions, may be mitigated by giving an active dose (for an adult ʒiij. of the bitartrate and fifteen or twenty grains of jalap corrected with a little ginger) early in the morning twice or thrice a week. The operation of the medicine passes over in a few hours, and the patient has leisure to recruit himself in the intervals between the doses. Christison speaks in high terms of gamboge which he employed in doses of five to seven or, very rarely, nine grains finely pulverized, with half a drachm of the bitartrate to prevent griping, every second day, or in urgent circumstances every day. Colocynth, scammony, and elaterium have also been employed; no remedy is superior to the last when the serous accumulation is very threatening and immediate effects are demanded. It may be given in doses of one-sixth or one-fourth of a grain every three or four hours until free evacuations have been obtained. It must not be overlooked that exhausting diarrhœa sometimes occurs spontaneously in the later periods of the disease, and that the use of drastics has been known to originate this untoward symptom; their use should, therefore, be immediately desisted from, if the diarrhœa show signs of proving intractable.

Warm baths are unquestionably the most effective of diaphoretics —they not only promote cutaneous transpiration, but often increase the secretion of urine at the same time. They may be applied in all their various modifications — warm water, hot air, steam, or the blanket-bath. Liebermeister describes as highly effective a method of applying the warm-water bath, by which the temperature, which is 98° when the patient first enters the bath, is gradually raised by the admission of warm water to 108°; after remaining in the bath about thirty-five minutes, the patient is packed in hot blankets. Headache, suffusion of the face, and unwonted heat of skin occasionally follow the use of warm baths, and may even necessitate their abandonment; generally, however, after a few trials patients can take their baths with pleasure as well as with advantage. The effect of pharmaceutical diaphoretics is very uncertain; the chief are Dover's powder, James's powder, and liq. ammon. acetatis.

Diuretics of the most opposite classes have been recommended by different writers; my own experience has not given me a high opinion of their efficacy. In judging of their effects, some observers have not sufficiently considered that a spontaneous diuresis is the normal issue of acute renal dropsy tending to recovery; and that in patients with contracting kidneys profuse diuresis is an ordinary feature of the quiescent state in the middle periods of the disorder: hence the conflicting testimony as to their utility. Bright, who had but slight confidence in them, was in the habit of prescribing uva ursi and pyrola umbellata; Christison relied on digitalis combined with cream of tartar. Rayer perceived little advantage from digitalis or squills, and found that at length they almost always deranged the stomach; according to his experience, horse-radish tea of all diuretics offered the best chance of success. Tincture of cantharides was employed by Dr. Wells in seven cases in doses of thirty to sixty drops a day, with good effect in five. Rayer also reports well of it in some cases, but thinks it "an uncertain remedy, which might be dangerous in inexperienced hands." I have tried in my own practice dandelion, broom-tops, and belladonna with unsatisfactory results.

When other means of evacuating the dropsical effusions fail, and erythema and gangrene of the skin of the lower limbs or genitals are imminent from the tension of the integuments, acupuncture or incision of the legs may be resorted to. The great disadvantage of this method is the liability that it may be followed by erysipelas unless stringent precautions are taken. I have tried both incisions and punctures and prefer incisions to punctures. One or two cuts with a lancet should be made lengthwise in the calf of the leg, or over the external malleoli, or one may be on the dorsum of the foot; they should be three-quarters of an inch long, and penetrate fairly into the subcutaneous tissue. The limb should then be wrapped in hot, moist flannels which should be changed at first every two hours; at each change the legs and especially the incised parts should be thoroughly sponged with warm water, and the flannels which are soaked with the discharge should be completely cleansed before being re-applied. Traube recommends that the incisions be frequently washed with chlorine water. If these precautions be adopted erysipelas will rarely follow. This treatment is followed by great relief at the time, and sometimes with prolonged advantage.

In cases characterised by a copious flow of urine (contracting kidney) dropsical effusions, if present, are usually slight and partial; and their existence depends chiefly on the watery state of the blood and the lowered tonicity of the tissues. In these cases ferruginous preparations, tonics, and mineral acids, by improving the general health, are more effectual against œdema than diuretics and cathartics. It is in cases of this class also that change of air or even a sea-voyage may be recommended, provided always that the disease be not too far advanced.

Bronchial catarrh and secondary inflammations should be treated with the primary mischief kept in view. Mercury and blood-letting are inadmissible—the former (unless in the most guarded way) on account of the peculiar susceptibility of the system in Bright's disease to mercurial preparations, the latter on account of the sapping of the strength which has already taken place. Internal antiphlogistics—aconite, digitalis, antimony—may be freely used together with the external application of chloroform epithems, hot poultices, and dry-cupping.

Dyspeptic symptoms are readily controlled in the early stages by a careful revision of the diet and the use of vegetable bitters, prussic acid, and antacids. When obstinate vomiting of uræmic origin sets in, creosote, morphia, and ice permitted to melt in the mouth are the most effective remedies. Diarrhœa of similar origin must be combated by acetate of lead, opium, and sulphuric acid.

Uræmic symptoms.—When these appear, renewed efforts should be made to increase the flow of urine, and to awaken the vicarious activity of the skin and intestines by the measures already described. If coma and convulsions have actually seized the patient, further energetic action is demanded. Frerichs, consistently with his view that carbonate of ammonia is the poison in these cases, recommends a treatment designed to neutralise the free ammonia and reduce it to a state of innocuous combination. He directs chlorine (in the form of inhalation or as solution) and the vegetable acids to be taken internally, the body to be sponged with vinegar, and vinegar to be used in injections. Chloroform inhalation is the most prompt and ready means of controlling the convulsive paroxysms; but when the uræmic seizure begins with drowsiness and gradually passes on into insensibility, or when convulsions occur only as breaks in a continuously comatose condition, chloroform affords no prospect of relief.

Blood-letting is a powerful means of staving off the immediate danger in uræmic coma, and convulsions; but it is only advisable in sthenic and recent cases. In the uræmic coma of acute Bright's disease, and in certain cases of puerperal eclampsia, the blood is as yet not materially impoverished, and the type of renal mischief is one that gives full hope of eventual recovery, while the attack itself is of extreme danger. In these, free and even repeated venesection is decidedly and urgently demanded. But it is otherwise when the renal mischief is chronic and incurable. The attacks themselves are not so imminently dangerous as when occurring in the acute form of the disease; patients frequently survive repeated uræmic paroxysms without the aid of venesection; the blood is commonly thin and poor; and lastly, there is not any prospect of ultimate recovery. Under these circumstances loss of blood is more likely to shorten than to lengthen life. Further, as Christison remarks, speaking of advanced renal mischief, when the torpor becomes considerable the removal of blood seems of little or no use. In some of the cases reported by Bright, death occurred from coma on the very day of free and repeated venesection. I can only conceive of two contingencies in which withdrawal of blood in quantity is justifiable in chronic renal disease; one is when coma comes on rapidly in a person whose constitution is not, as yet, seriously deteriorated, and whose prospects of life, apart from the uræmia, may extend to many months or some years; the other is, when there is a necessity for temporary restoration of the faculties paramount to the general chance of prolonging life.

NEPHRITIS AND PYELITIS CONSECUTIVE TO AFFECTIONS OF THE LOWER URINARY TRACT.

BY MARCUS BECK, M.S.

THE secondary affections of the kidney arising from diseases of the lower urinary tract have perhaps received less attention from physicians than their great importance and frequency would deserve. It is for this reason partly that their exact diagnosis and pathology are still but imperfectly understood. Their frequency may be estimated from the fact that of sixty-six deaths from diseases of the urinary organs occurring in the surgical wards of University College Hospital, in a period of six years, forty were directly due to secondary inflammation of the kidneys. In the practice of the physician it is less common to meet with cases in which secondary renal inflammation forms an important element of the disease. It is, however, a frequent termination of the cystitis occurring as a consequence of retention and decomposition of urine in those diseases of the spinal cord and brain which give rise to paralysis of the bladder, and in cases of calculous pyelitis the changes in the substance of the kidney are identical with those resulting from many diseases of the bladder.

The morbid conditions of the kidney arising as the consequence of vesical or urethral disease, may be divided into four chief groups.

1st. The effects of pressure caused by the obstruction in the urinary passages.

2nd. Diffuse interstitial inflammation, acute or sub-acute.

3rd. Interstitial inflammation with scattered points of suppuration.

4th. The cicatricial kidney, occurring after recovery from diffuse or localized interstitial inflammation.

Although typical specimens of these four varieties are readily recognisable and distinguishable from each other, it is most common to find them variously intermixed. Thus a kidney showing in a high degree the effects of pressure may present, in addition, either acute diffuse inflammation or scattered points of suppuration, and in the same way a small puckered cicatricial kidney may suffer from a fresh acute attack from a recurrence of the primary disease in the lower urinary tract.

In the great majority of cases the final stages of the disease are accompanied by acute pyelitis, and it was on this account that Rayer gave the name of pyelo-nephritis to this affection. Pyelitis is not, however, a constant accompaniment even of the acute suppurative form.

I. THE EFFECTS OF PRESSURE.

Causes of Obstruction to the Flow of Urine.—In any obstruction of the urinary passages, the secretion of urine still continuing to take place, the parts above the obstruction become distended and tensely stretched. If the obstruction be complete, as soon as the pressure in the urinary passages becomes equal to the blood-pressure in the vessels of the kidney, secretion will be to a great extent arrested. Such a condition therefore does not lead to rupture of the ureters or bladder unless their coats are weakened by previous disease. The obstruction to the free flow of urine may exist at any part of the urinary tract from the pelvis of the kidney to the orifice of the urethra, and in some rare cases it is said that a tight phimosis has given rise to serious secondary renal disease. The effect upon the kidney is not however induced in exactly the same way in all cases. In the ureter the most common cause of complete obstruction is the impaction of a calculus or some congenital malformation. Incomplete obstruction is caused occasionally by tubercular deposit in the submucous tissue, and by pressure from tumours. The most common seat of obstruction is, however, at the entrance of the ureter into the bladder, as by the growth of a villous tumour round about the orifice. In the above cases the cause and nature of the obstruction are evident. It is not quite so clear, however, how it is that the same signs of obstruction to the flow of urine from the kidney are found in most cases of neglected stone in the bladder or stricture of the urethra. It has been supposed that the natural valved arrangement of the entrance of the ureter into the bladder becomes deranged, so that at each act of micturition some urine is forcibly driven backwards into the kidney. This theory is, however, untenable; in the first place because intermittent pressure, occurring at such long intervals, would not cause the amount of dilatation of the kidney often found in these cases; and secondly, because numerous observations have clearly shown that the valvular action of the orifices of the ureters is scarcely ever, if ever, impaired. We must therefore look for some obstruction to the flow of urine at the orifices of the ureters themselves, and there is no difficulty in finding enough to account for the effects produced. In all cases of stone, or stricture of the urethra, in which great signs of pressure are found in the kidneys, two conditions are almost constantly present, hypertrophy of the bladder, and chronic inflammation, with thickening of the mucous membrane. In a hypertrophied bladder the thick bundles of muscular tissue will press more or less into the ureter and tend to narrow its calibre; the swelling of the orifice and the thickening and induration of the mucous membrane and submucous tissue still further tend to narrow the passage for the urine; and thus a more or less tight stricture of the orifice is produced, obstructing the free flow into the bladder, and necessitating a constant increase of pressure in the kidney itself in order to drive the urine onwards. In enlargement of the prostate, the bladder can never be completely emptied. It is always kept at a certain degree of tension, and of course the same pressure must exist in the parts above, even to the very ends of the urinary tubules. Similar signs of pressure in the kidney are also found occasionally as the result of simple chronic cystitis. Under these circumstances it is due, as in stone, to hypertrophy of the bladder and thickening of the mucous membrane at the orifice of the ureter. If any further proof were needed that it is at the orifices of the ureters, and not at the neck of the bladder, or in the urethra, that we are to look for the immediate cause of the obstructed flow from the kidney in cases of stone, stricture, or cystitis, it is furnished by the fact that the signs of pressure are often very different on the two sides, showing that we must look for something more than the single obstruction in front to account for the changes in the kidney.

The effects of long-continued increase of Urinary Pressure as the result of Obstruction to the Passage of Urine.—The observation of the uncomplicated effects of increase of the urinary pressure in the kidney is possible in only a small proportion of cases. In the great majority the fatal result is brought about by an acute attack of inflammation, usually accompanied by the extension of decomposition of the urine from the bladder to the pelvis of the kidney, and consequent acute pyelitis and suppurative nephritis; and thus the chronic disease which preceded the acute attack is completely concealed. It is only in cases of pressure upon the ureter from without, and in a few of disease of the bladder or urethra, in which death occurs from some independent cause, that the effect of increased pressure can be studied with advantage.

The earliest effect observable is a slight dilatation of the ureter and pelvis of the kidney. The walls of the ureter are thickened, chiefly from hypertrophy of the muscular coat. The mucous membrane is usually of a more opaque white colour than natural. The calices of the pelvis are somewhat dilated. The kidney itself is slightly increased in size. The capsule strips off without any particular difficulty, but may tear the kidney substance in so doing. The venous stars on the surface are usually well marked, and the larger veins of the kidney are often full of blood. A section of the kidney shows the cortex pale, slightly increased in width, and tough in consistence. The Malpighian bodies may be seen as red dots in some cases. The pyramids show little change; they are usually pale like the cortex. Microscopic examination shows that the whole of the changes are due to an overgrowth of the interstitial connective tissue of the kidney. This is usually most marked about the Malpighian bodies, which may be surrounded by a complete zone of small round cells, sometimes more than one layer in thickness. The change is extremely irregular, varying in every field of the microscope; parts which are apparently perfectly healthy existing side by side with others in which the change is very marked. The epithelium shows little, if any, change. In the pyramids the straight tubules may be slightly dilated.

This form of kidney is somewhat difficult to recognise without the aid of the microscope, as from the anæmic appearance of the cortex and the increase in size, it may resemble the effects of catarrhal inflammation or cloudy swelling. The anæmic appearance of the kidney after death must not be taken as an indication that it was in the same condition during life. In all renal inflammations, accompanied by swelling or by increase of the fluid pressure in the urinary tubules, the distension of the organ is resisted to a certain extent by the capsule, and all the tissues of the organ are more or less squeezed together. The consequence of this is, that as soon as the action of the heart ceases, the vessels empty themselves, partly backwards into the renal artery, and partly onwards into the veins. It is for this reason that the larger veins are sometimes full of blood.

The next stage of the effects of pressure is characterised by a gradual absorption of the pyramidal portion of the kidney. This commences from the papillæ, which at first become rounded and flattened. Ultimately the whole pyramidal portion of the kidney may entirely disappear, the situation of each pyramid being marked by a deep hollow, formed by the expanded calyx. This process of destruction of the pyramids is one of absorption, not of ulceration. The hollow left is lined with a perfectly smooth, white membrane, continuous with the mucous membrane of the pelvis. The cortex may for some time after the destruction of the pyramids remain thicker than natural from the great overgrowth of the interstitial connective tissue. Finally it also becomes stretched and thinned, till the whole kidney may resemble a large, thick-walled cyst; the wall on one side being composed of the thickened and expanded pelvis, and on the other of the stretched and thinned cortex. This form is usually spoken of as the sacculated kidney. In these cases the capsule is thickened and firmly adherent. It may be more adherent than natural to the surrounding fat. The cortical substance is exceedingly tough, so much so that it cannot be broken down by the pressure of the finger and thumb. Its colour is opaque and whitish. The ureter may be dilated to such an extent as to resemble a piece of the small intestine. The microscope shows that the change is accompanied by great overgrowth of the intertubular connective tissue—a condition of chronic interstitial inflammation. The new growth is very abundant round the Malpighian bodies, and may accumulate to such an extent as to strangulate the vessels and cause their obliteration. The corpuscle is then composed of a central transparent substance, almost homogeneous in appearance, but marked by a few concentric curved lines representing the obliterated tufts of vessels, surrounded by a concentrically-laminated layer formed from the thickened capsule. In other bodies the capsule may be greatly thickened without the vessels being obliterated. This thickening of the Malpighian capsule occurs very early in the affection, and when found in kidneys in which an acute attack of interstitial inflammation has immediately preceded death, it forms a valuable indication of the antecedent chronic affection. The tubules are usually irregularly dilated in parts, but the dilatation is not so great as might be expected, being limited by the new growth between the tubules. If any of the pyramidal portion remain, microscopic examination shows merely dilatation of the straight tubules, with abundant small-cell infiltration of the intertubular structure. The epithelium throughout the kidney is healthy.

Complete obliteration of the ureter does not arise as a secondary result of diseases of the bladder or urethra. When it does occur it is usually due to congenital malformation, pressure of tumours, impaction of a calculus, or tubercular pyelitis, and it will be treated of with these diseases.

The effects of increase of pressure in the urinary tubules may therefore be said to be the induction of a chronic interstitial nephritis, accompanied by the gradual absorption of the structures of the kidney, commencing with the pyramids and gradually extending to the cortical substance. That abnormal pressure in the urinary tubules should thus induce a chronic interstitial change is easily explained. Undue distension of the urinary tubules cannot exist without a greater or less degree of obstruction to the circulation through the vessels of the kidney, acting most forcibly on the veins, as in them the blood-pressure is lowest. The venous flow thus being more or less impeded, a condition of passive congestion will be induced, and it is well known that from whatever cause this arises, its most marked effect on the kidney is an overgrowth of the connective tissue. This alone, however, would not account for all the changes resulting from obstruction in the urinary passages, as it is not merely an overgrowth of the connective-tissue that is found, but an accumulation of small round cells far exceeding in amount that arising merely as the result of passive congestion, and this we must look upon as a chronic inflammatory change set up by the direct irritation of the tension to which the kidney substance is exposed.

The overgrowth of the connective tissue of the kidney which results from an increased pressure in the tubules may be looked upon partly as a beneficial change, rendering the kidney more capable of withstanding the stretching to which it is exposed; and it is for this reason that, even in very advanced cases, the dilatation of the tubules is seldom very great. Moreover, as the secretion of urine is in a great measure dependent upon the difference between the pressure of the blood in the Malpighian tufts and that of the urine in the tubules, it would be diminished to a dangerous degree, were not that in the Malpighian tufts somewhat increased by the slight obstruction to the venous circulation caused by the new growth between the tubules. It is as a result of this that, so far from the secretion being diminished, it is usually increased, often greatly, and at the same time its specific gravity is correspondingly lowered. On the other hand, a kidney, the venous circulation of which is more or less obstructed, if exposed to an attack of acute congestion such as may arise from exposure to cold, or follow the introduction of a catheter, suffers much more severely than a healthy organ. Its vessels may become choked with blood to such an extent as to lead to complete stasis, accompanied necessarily by cessation of the secretion of urine, which unless shortly relieved must terminate fatally. This seems to be the explanation of many of those cases of death from suppression of urine which follow an operation on the bladder or urethra. In other cases the acute hyperæmia may not be sufficient to cause complete stasis, but as the kidney is already suffering from a chronic inflammatory change, it may exaggerate the activity of the process, and convert the chronic interstitial nephritis into an acute form, causing such injury to the organ as speedily to prove fatal.

Supposing the obstruction to the escape of urine to be completely relieved, as by the regular introduction of a catheter in enlarged prostate, or by the cure of a stricture, the chronic cystitis will subside, and the bladder, if it have been hypertrophied, will gradually assume its normal proportions, and thus all impediment to the free flow of urine through the orifices of the ureters will be removed. The cause of irritation to the kidney being thus removed, a process of cicatricial contraction of the new growth between the tubules sets in, and the form afterwards to be described as the cicatricial kidney is produced.

In no case that I have observed has the chronic interstitial nephritis arising from increased urinary pressure, even when it was due to great contraction of the organ, been accompanied by the hypertrophy of the heart so commonly seen with the granular contracted kidney; and as the histological morbid conditions are almost identical, this fact would support the view of Sir W. Gull and Dr. Sutton, that the hypertrophy of the heart is due to a general change in the small arteries and capillaries, and not to the condition of the kidney. The small arteries frequently show great hypertrophy of their muscular coats, such as is seen in the granular contracted kidney, but this would seem to be due merely to the increased tension to which they are exposed in consequence of the obstruction to the venous flow. Such increase of tension must call for greater force in the muscular coat of the artery, in order that it may perform its function of regulating the blood-supply, and hence the hypertrophy; for it is an invariable law that when involuntary muscular fibre is called upon for increased work, hypertrophy speedily results.

2. DIFFUSE INTERSTITIAL NEPHRITIS.

In this form of secondary renal inflammation both kidneys are invariably affected. The whole kidney is swollen; the surrounding fat is occasionally somewhat adherent to the capsule, but usually separable without great difficulty. The capsule is less transparent than natural, and may be abnormally vascular; small vessels, visible to the naked eye, ramifying in its structure. It separates with ease, but in so doing often slightly tears the kidney substance, and leaves the surface coarse and irregular. On removing the capsule, the kidney is seen to be of a pale yellowish-white colour, or it may be mottled here and there with red patches. If it present a mottled appearance, the red and white patches will be found often to be arranged so as to correspond with the bases of the various lobules of the gland, in some of which the disease is more advanced than in others, the pale parts being those in which the change is most extensive. The stellate veins usually to be seen on the surface of the kidney are in most cases very clearly marked and gorged with blood. On laying the kidney open the cut surfaces become slightly convex, showing that the elements of the kidney have been compressed within the capsule by the exudation between the tubules. The cortex is of a pale yellowish-white colour, or mottled with red, like the surface. The consistence varies; if the attack is of quite recent origin, and has not been preceded by any chronic change due to increase of pressure in the urinary tubules, it is very soft, breaking down readily under the pressure of the finger and thumb; on the other hand, if there has been a previous overgrowth of the interstitial or connective tissue, it may be tougher than natural. The Malpighian bodies are not usually clearly visible, but they may appear as red dots. The pyramids are sometimes pale, but not unfrequently intensely red, contrasting strongly with the pale cortex. Yellowish streaks may be seen in some cases running parallel to the straight tubules, but their appearance is by no means constant. The anæmic condition of the cortex, as it is seen after death, must not be taken as evidence that during life it was in the same condition. It is most probable that the vessels empty themselves as soon as the heart ceases to beat, the kidney being, as before stated, somewhat compressed within its capsule. Confirmatory of this is the fact that the large veins at the cortico-medullary junction and the interfascicular veins are usually fully distended with blood. The pelvis may in some cases be perfectly healthy; in others, its mucous membrane is congested, branching vessels ramifying in all directions in its structure; but most frequently it is inflamed, intensely reddened, and sometimes covered with a membranous exudation. The contents of the pelvis are sometimes acid and free from odour; and it is in these cases that there is little or no pyelitis. In others the urine is excessively foul, and has evidently been in a state of decomposition before death; and, under these circumstances, the intense inflammation of the pelvis is always present.

It is not always easy to recognise the exact condition of the kidney by the naked eye, as the appearances may closely resemble those of the large white, or the fatty kidney. The microscope alone can in all cases exactly determine the pathological changes. On examination of a thin section of the cortex, it will be found that the chief change noticeable is an abundant accumulation of small round cells between the tubules, and especially round about the Malpighian corpuscles. The condition exactly resembles that before described as resulting from simple increase of pressure in the urinary tubules, but greatly intensified. The accumulation of small round cells is not by any means uniform. In the same field of the microscope may be parts in which the kidney structure may appear almost normal, and others in which the tubules may be pressed upon by the new growth to such an extent as completely to close them. In parts in which the new cells are crowded together to this extent, it is often difficult to recognise the tubule. No trace of a membrana propria is to be seen, and the site of the tubule is marked merely by a group of epithelium cells packed closely together amongst the surrounding small round cells; the Malpighian bodies, besides being completely encircled by multitudes of new cells lying outside their capsules, usually present large numbers of nuclei crowded amongst their loops of vessels, so that they may be scarcely recognisable. The epithelium generally presents no very marked changes, beyond being somewhat more granular than natural, and a little swollen. The nuclei of the cells are clearly visible in specimens prepared with the ordinary staining fluids, and no reagents are necessary to bring them into view. Oil globules are never present. It is not uncommon to find some of the tubules plugged with fibrinous casts. In very acute cases small round cells, exactly resembling those outside the tubules, are found within them, surrounded by epithelium, which shows no marked signs of change beyond slight cloudiness and swelling. It is difficult to suppose that these are derived from the epithelium; and it is far more probable that they have found their way in from without. If these cells are washed on by the flow of urine from above they would appear as pus cells in the urine. The epithelium washes out of the tubules very readily, possibly on account of its adhesion to the membrana propria being lessened; but this must not be taken to indicate a condition of desquamative catarrh; nor must it be assumed that the tubules seen to be empty in the preparation, were empty during life. More gentle handling of the sections whilst mounting them would probably have enabled the observer to obtain specimens in which all the tubules were lined with epithelium. The blood-vessels are usually empty, but in some cases they may be found engorged with blood, and even in parts ruptured, giving rise to small extravasations in the inter-tubular tissue, and occasionally even into the tubules themselves.

3. Acute Interstitial Nephritis, with Scattered Points of Suppuration.

(Suppuration of the Kidney, suppurative nephritis; and, when accompanied by pyelitis, pyelo-nephritis.)

This form of kidney is found in the great majority of fatal affections of the lower urinary tract, and it has been wrongly assumed by some writers to be the only form of fatal renal affection occurring in such cases. It has also been stated that it is invariably associated with acute pyelitis and the presence of decomposing urine in the pelvis of the kidney. This also is incorrect. I have seen several cases of disseminated suppuration of the kidney, in which the pelvis was free from acute inflammation, and the urine it contained acid and without any odour of decomposition. In one case both kidneys were equally dotted with minute points of suppuration, but in the pelvis of one only was the urine decomposing.

The naked-eye appearances of acute suppurative nephritis consist essentially of those described as occurring in simple acute nephritis, with the addition of minute scattered abscesses, varying in size from a pin's head to a pea, almost invariably confined to the cortical substance. The fat surrounding the kidney is usually œdematous, and more or less adherent to the capsule. The whole kidney is enlarged, swollen, and softened. The capsule separates readily, often tearing the kidney substance in so doing. The act of stripping off the capsule often opens up many of the small collections of pus. Occasionally they are found to have burst beneath the capsule, which may be for a considerable area separated from the kidney substance by pus. If this be not the case, each collection of pus shows as a yellow spot on the surface of the kidney, surrounded in most cases by a zone of intense red, due to congestion. In an earlier stage yellow spots, surrounded by a red zone, are often seen, which, on being cut into, are found not to contain pus, but to be excessively soft, and on the point of breaking down into fluid. Between the points of suppuration the surface of the kidney is irregular and coarse, and of a pale yellowish-white colour, often mottled with red, as in simple acute interstitial nephritis. The small abscesses are usually grouped together in areas corresponding to the bases of the lobes of the kidney; that is to say, the areas corresponding to separate pyramids of Malpighi. Some of these areas are completely free, whilst others contain many minute points of suppuration. The exact situation of the individual abscesses is more difficult to determine from an observation of the surface. Rindfleisch states that they correspond to the points at which the interfascicular veins appear upon the surface. It must not be concluded, however, that because they appear in this situation the vein has anything directly to do with their production, for it is at this point that the connective tissue is most abundant, and the lymphatics of the kidney follow the same course as the veins. On bisecting the kidney, the cortex presents a mottled appearance, similar to the surface; but the chief point to be observed is the relation of the small purulent collections. These will be found to be almost always continued downwards in the form of pale yellow streaks, extending into the cortical substance, along the course of the interfascicular veins, and continued in many cases into the pyramids. These pale streaks follow, therefore, the course of the connective tissue and lymphatics, which are most abundant in the neighbourhood of the interfascicular veins. The abscesses, and the pale streaks between them, naturally assume a wedge-like form, in consequence of the anatomical arrangement of the structures amongst which they are situated; but this is very different from the form of an embolic abscess. In embolic patches the width of the base of the wedge is seldom less than half its length, but in these abscesses of interstitial nephritis the base showing on the surface may be only the width of a pin's head, while the length of the wedge may be one inch or more. Embolic patches, moreover, scarcely ever extend into the pyramids.

The medullary portion of the kidney is often intensely congested, but more commonly it is mottled with yellowish-white streaks, running parallel to the tubules. Dickinson describes "sharply defined white lines which start from the tops of the mammillary processes in the pelvic cavity, and pass into or through the cones in the direction of their striation." These he states to be dilated tubes (Med.-Chir. Trans., 1873). Such lines are no doubt occasionally visible, but by no means constantly so.

The pelvis of the kidney is in the great majority of cases acutely inflamed. Its mucous membrane presents usually patches of dark grey pigmentation, the result of previous attacks of pyelitis, but the greater part is of an intense purple. Here and there may be seen patches of ulceration, and the surface may be covered by a dense false membrane, like that of diphtheria, which is usually gritty, from the deposit of amorphous phosphates in and upon it. The mucous membrane is everywhere swollen and softened. The whole ureter is usually in a condition similar to that of the pelvis. In the cavity of the pelvis is usually an abominably fetid mixture of putrid urine, mucus, and pus, often tinged with blood. Acute pyelitis is, however, not a constant accompaniment of suppurative nephritis. Cases do occasionally occur in which the ureter and pelvis are quite free from acute inflammation.

A microscopic examination of a kidney in a state of acute suppurative inflammation shows clearly that the change does not commence within the tubules. It is primarily extra-tubular, and the alterations in the epithelium, which are the same as those before described, are evidently secondary to the interstitial change. All the morbid changes previously described, as occurring in acute interstitial nephritis without abscesses, are to be seen in this form of the disease also, but at certain points the inflammatory process has reached the stage of suppuration. In a pyæmic kidney the disease is localised around the spots at which the septic embola have lodged, and the rest of the kidney is healthy, with the exception of the swelling of the epithelium, invariably found as the result of pyæmia or septicæmia. In acute suppurative nephritis, the change is much more general, no large area of the kidney being found free from marked interstitial change; but, as in the non-suppurative form, the degree of affection varies greatly in almost every field of the microscope. If a section be cut so as to include the edge of one of the small points of suppuration, the following conditions are seen, proceeding from without inwards, towards the centre of the abscess. Most distant from the centre the only sign of disease will be an accumulation of small round cells between the tubules. The epithelium is usually slightly swollen and cloudy, but the lumen of the tubule is quite free, and the nuclei of the cells are readily seen without the use of reagents. The outline of the tubule is distinctly visible. In the next stage nothing resembling a membrana propria is to be seen. The epithelium cells are pushed together and are closely surrounded on all sides by the small round cells. Frequently the tubule is seen to be filled with small round cells similar to those outside it: the epithelium still being distinctly recognisable. Nothing is seen in the epithelium cells to suggest that these small round cells have been derived from them. Finally all trace of tubule and epithelium disappears, and the space becomes occupied merely by a mass of small round cells, which after the softening of the intercellular substance becomes pus. The vessels in the affected area vary much in appearance. In some cases they seem to be emptied of blood immediately after death, and they are then very difficult to recognise; in others they are greatly engorged. It is not unfrequent to find clots in some of the smaller veins, both in the pyramids and cortex, but this would seem most probably to be a secondary effect of the inflammation going on outside them. Of the lymphatics it is impossible to speak positively, as, in addition to the great difficulty at all times existing in examining these vessels in the kidney, they are completely obscured in acute interstitial nephritis, by the abundant small cell infiltration of the intertubular tissue. It must be remembered, however, that the chief seats of inflammation and suppuration are in most cases along the lines of the interfascicular veins, and in this situation the lymphatics are most abundant. In the pyramids the straight tubules are almost invariably denuded of epithelium.

In cases in which the acute inflammation has followed on a chronic process, the signs of the previous disease are usually completely concealed by the recent change. Occasionally, obliterated Malpighian tufts may be found, which are certain evidence of the antecedent chronic change.

In conclusion, it may once more be stated that the suppurating kidney, secondary to affections of the lower genito-urinary tract, is not merely an affection in which scattered points of suppuration occur in the substance of the gland, but it is an acute interstitial inflammation, affecting the whole kidney, and culminating at various points in suppuration.

In acute diffuse interstitial nephritis, whether suppurative or not, the condition is very frequently conjoined with signs of previous distension of the kidney by increased pressure within the urinary tubules. The pyramids may be more or less completely destroyed, the cortex even expanded and thinned, and the pelvis and ureters greatly dilated. There is no doubt that the acute forms, whether suppurative or not, are greatly predisposed to by the chronic interstitial change set up by the distension of the kidney.

As an occasional result of suppurative nephritis, an abscess may burst through the capsule, and give rise to a large collection of pus round about the kidney (peri-nephritic abscess). Similar collections of pus may form rapidly from ulceration and perforation of the pelvis, especially if the ureter become blocked from any cause. That the formation of a peri-nephritic abscess is so rare in suppurative nephritis, is due to the fact that it is rare to find only one kidney affected, and when both are attacked death usually supervenes, long before the abscesses reach such a size as to burst through the capsule.

4. The Cicatricial Kidney.

It is not at all unfrequent in making post-mortem examinations of patients, who have at some time suffered from an affection of the urinary organs, to find one or both kidneys shrunken, toughened and irregular in form. This condition may so closely resemble the ordinary granular contracted kidney, as to be indistinguishable from it. In fact, as both are the result of an overgrowth and cicatricial contraction of the interstitial tissue of the kidney, it is only to be expected that their appearances should be the same. In other cases the distinction can be readily made by the signs of the abnormal urinary pressure, which has at some time existed, such as a more or less complete destruction of the pyramids. If the cause of the urinary obstruction have been long removed, the pelvis and ureter, if they have been previously dilated, return to their normal size, so that their appearance is of little value. The mucous membrane may, however, be more or less pigmented, as the effect of previous pyelitis. Occasionally, distinct cicatrices are found on the surface of the kidney and in the capsule. It has been suggested that these are scars of former abscesses which have discharged themselves by the tubules. The evidence of this is not, however, perfect, for a cicatrix of an exactly similar kind would be left if the inflammatory process had stopped at a point at which considerable destruction of the kidney tissue had occurred, but the inflammatory products had not actually broken down into pus. In these cases of cicatricial kidney, the capsule is often found to be very firmly adherent to the surrounding fat. A kidney which has thus healed may become the seat of a fresh acute attack from a recurrence of the original disease of the lower urinary passages; and, then, with the form of a contracted kidney are combined the colour and other appearances of acute interstitial nephritis.

Microscopic examination of a cicatricial kidney shows merely a great excess of connective tissue between the tubules, which are in some places narrowed by the pressure of the new growth, and in others dilated in consequence of the obstruction at a lower point. Many obliterated Malpighian corpuscles are usually to be found in all parts of the cortex.

ETIOLOGY OF SECONDARY INTERSTITIAL NEPHRITIS.—Three chief causes are at work in various degrees in the production of secondary renal disease. 1. Increased pressure in the tubules from obstruction to the escape of urine. 2. Reflex irritation of the kidney. 3. The presence of septic matter in the pelvis of the kidney, and possibly in the lower parts of the tubules. All these three causes may be acting together, as is by far the most frequent case, or one may act singly. It may be broadly stated, however, that either of the first two—acting alone—rarely if ever gives rise to suppuration, although it is probable that in combination they do so. At least, cases have come under my observation in which it was difficult to find any other explanation. The last, acting alone, is doubtless capable of inducing suppuration rapidly, but it does so more easily when the kidney is already suffering from the effects of the other two.

1. *Increased Urinary Pressure.*—The effect of this in causing chronic congestion and interstitial nephritis has already been discussed.

2. *Reflex Irritation of the Kidney.*—In order to justify the insertion of reflex irritation amongst the causes of secondary renal inflammation, it is only necessary to remind the reader of those cases in which the simple passage of a catheter has been followed by complete suppression of urine and death, and of those, in which a patient suffering from some urinary affection, involving obstruction to the escape and decomposition of the secretion, has died in a few days after some simple operation, such as the introduction of a sound or lithotrite, or the passage of a bougie, acute inflammation of the kidneys being found. It is impossible in these cases not to conclude that the passage of the instrument is the direct cause of the exacerbation of the renal inflammation and of the fatal result. All other conditions remain unaltered; the passage of the instrument is the only new element introduced. In such a case also it is difficult to conceive that the introduction of septic matter on the instruments employed can have anything to do with the result, as the urine itself was before in a state of decomposition.[1] Observations have convinced me that

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[1] In order to obtain, if possible, some definite evidence of the effect upon the kidney of operations upon the urethra, I examined carefully the urine passed, as to quantity, specific gravity, blood, and the amount of urea excreted, in four cases—three of

the appearance of blood in the urine some hours after a severe operation upon the urethra, with an increased quantity of urine, is not by any means an uncommon occurrence. The latter may sometimes be preceded by a temporary suppression. These facts point very strongly to the occurrence of a dilatation of the renal vessels, and an active congestion of the kidney, preceded probably by a contraction of the vessels and anæmia of the kidney of varying duration, in some cases so transient as hardly to be noticeable, in others sufficiently prolonged to be recognisable by the temporary cessation of the flow of urine. No conclusions could be drawn from the estimation of the urea, except that in all probability the disturbance in the kidney is purely vascular, and does not extend to the proper tissue of the gland. If such a

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dilatation of a stricture by Holt's dilator, and one of internal urethrotomy. In each case a catheter was tied in after the operation and the urine drawn off at intervals. In the first case, one of very old stricture, the patient passed only 15 cc. of urine in the first three hours after the operation. Its specific gravity could not be taken. The amount of urea it contained was 3·15 grms. It was clean and quite free from blood. In the following three hours 225 cc. of urine, of a specific gravity of 1·024, was passed. It was darkly tinged with blood; the amount of urea was 4·725 grms. In the next three hours 160 cc. were passed still tinged with blood, spec. grav. 1020. After this the quantity of urine became less, and the blood completely disappeared in twenty hours. In the second case, also of an old stricture, 250 cc. were passed in the first three hours, darkly tinged with blood. The specific gravity was 1033. The amount of urea 3·75 grms. In the next three hours the quantity fell to 190 cc., and after that there was nothing to note as to amount excreted. It remained slightly tinged with blood, uniformly mixed and free from clots, for 24 hours. From the fifteenth to the eighteenth hour the blood almost disappeared, but it returned in the next three hours; and during that time his temperature rose slightly from 99·2 Fahr. to 100·2 Fahr. In the third case—of Holt's dilatation—the quantity showed scarcely any variation. In the first hour 34 cc., in the second 34 cc., and in the third 30 cc.; and in the next three hours 93 cc. of urine passed. The specific gravity was 1020. The only point of interest in this case was that although there was scarcely any bleeding at the operation, and the urine for the first hour was almost free from blood, yet the quantity steadily increased up to the end of the third hour, and disappeared at the end of the sixth hour. It was uniformly mixed with the urine, and free from clots. The fourth case—one of internal urethrotomy—was a young man who afterwards unfortunately died of pyæmia. The kidneys at the post-mortem examination were found to present scarcely any signs of disease. In this case, in the first hour the patient passed 260 cc. of pale urine, of a specific gravity of 1003, and containing 1·56 grms. of urea. In the next two hours he passed 150 cc. of a specific gravity of 1012, and containing 2·70 grms. of urea. In the next hour only 42 cc. of urine were passed, and the same hourly rate was maintained till the patient went to sleep. The urine passed in the first hour was almost free from blood; in the second and third hours the blood increased, and up to the tenth hour blood was present in sufficient quantity to render the urine dark-red, after which it diminished and had completely disappeared by the sixteenth hour. In three of these cases the quantity of urine passed was markedly increased, in one very remarkably so during the first few hours after the operation; and in one of these the increase was preceded by a period of almost total suppression. In the remaining case but little alteration was observed. In every case the amount of blood steadily increased for the first few hours, and then gradually subsided. In the first case, during the period of almost total suppression, the urine was free from blood, but as the quantity increased the blood made its appearance. Was this blood from the kidney, or was it from the wound? It seems difficult to conceive that a lacerated wound, such as is produced by the forcible rupture of a stricture, could continue to bleed for so long a time. If the blood were from the wound it is most probable that some clots would have been seen; and if it were slowly trickling back into the bladder along the outside of the catheter, it would not have been uniformly mixed with the urine. Now in all these cases no clots were seen, and the blood was uniformly mixed with the urine. It seems most probable, therefore, that the blood in these cases actually came from the kidney.

condition of active hyperæmia be induced in a kidney already suffering from a sub-acute or chronic interstitial inflammation as the result of over-distension, it is easy to conceive that it may hasten the inflammatory process, and the sub-acute may become acute inflammation, possibly even reaching the stage of suppuration.

The trigone of the bladder and the prostatic and bulbous portions of the urethra seem to be the parts the irritation of which most directly affects the kidney; for it is extremely rare for any operation upon the penile portion of the urethra to be followed by any acute renal inflammation. These parts are very abundantly supplied with nerves, both from the sympathetic and cerebro-spinal systems. The sympathetic branches come from the hypogastric plexus, which is a direct continuation of the aortic, and the aortic is derived from the solar plexus, which also furnishes a great part of the renal plexus. There is no difficulty, therefore, in conceiving that any stimulation applied to the bladder or urethra may react directly on the kidney, causing a temporary contraction, followed by a more or less prolonged dilatation of the renal vessels. Stimulation of the sensory nerves of the bladder, which are derived from the communicating branches from the sacral spinal nerves to the pelvic plexus of the sympathetic, would also cause a dilatation of the renal vessels, but without any antecedent contraction. In stone in the bladder, and in stricture of the urethra, every time the patient passes water a more or less violent stimulation of the nerves of the bladder or urethra necessarily occurs, and it is probable that the stimulation is accompanied by a corresponding vascular disturbance of the kidney. These repeated attacks of hyperæmia, although they would be comparatively harmless in a healthy kidney, tend to aggravate any inflammatory changes that may have been set up from the other causes mentioned above.

3. *The presence of Septic Matter in the Pelvis of the Kidney.*—In the great majority of cases the presence of decomposing urine in the pelvis of the kidney is the immediate cause of the final acute attack, which terminates the chronic nephritis secondary to vesical or urethral disease; but it is an error to suppose that the presence of ammoniacal urine in the pelvis is an essential cause of suppuration in the kidney. Cases do undoubtedly occur in which suppuration occurs with acid urine in the pelvis. I have seen several myself during the past few years. It is not enough to examine the urine as passed by the patient before death, for alkalinity of the urine as it is discharged from the bladder is fortunately only accompanied by extension of the decomposition to that in the ureters and pelvis in a comparatively small proportion of cases. This is easily proved by washing out the bladder carefully, until the water that returns by the catheter is perfectly clean. Then, if the instrument be tied in for a short time, the first urine that comes away will almost always be acid. Should it be alkaline from the presence of ammonia the condition of the patient is a most serious one, as it indicates the presence of putrid urine in the pelvis of the kidney. The extension of the process of

decomposition to the urine contained in the ureters is prevented, probably, by the almost constant flow of fresh urine through their narrow orifices into the bladder. This stream washes back the solid particles (whether organisms or not has no bearing on the present question,) which are the cause of the ammoniacal fermentation of urine. It has before been stated that regurgitation of urine during micturition is a very rare occurrence, if it ever takes place; and some other explanation must therefore be found for the extension of decomposition from the bladder to the ureters. The most probable supposition is, that in consequence of the irritation to which the pelvis and the ureters are exposed in common with the kidney, from the obstruction to the free passage of the urine, a slight degree of inflammation is set up, accompanied by an increase in the secretion of mucus. If, then, the ureter, instead of having its mucous surface constantly clean and washed by the passing urine, is covered with a thin layer of almost stagnant mucus, it is easy to see how decomposition may spread upwards from the bladder. It is possible, also, that the catarrhal inflammation existing in the bladder may extend into the ureters by direct continuity of tissue; but this is certainly a rare occurrence, for when the opportunity occurs of examining a case of catarrhal inflammation of the bladder, in which the patient has died from some other cause than suppurative nephritis, the redness and swelling are found almost invariably not to extend beyond the orifice of the ureter. It is therefore probable that in all cases slight catarrhal pyelitis precedes the decomposition of the urine in the pelvis of the kidney; but as soon as the decomposition occurs, the pyelitis becomes greatly intensified by the irritation of the ammoniacal urine.

The exact mode in which the decomposing urine affects the kidney so as to give rise to a degree of acute inflammation incompatible with life, is a question upon which there exists much difference of opinion.

If the kidney be already suffering sub-acute interstitial inflammation from the other causes already mentioned, it is evident that the increased irritation set up by the presence of ammoniacal urine filling the pelvis, and giving rise to acute pyelitis, would be quite sufficient to intensify the process to such an extent as to render the kidney incapable of performing its functions. Suppurative nephritis is, however, frequently observed in kidneys in which the signs of previous disease are almost or wholly wanting, and in these cases the acute inflammation is not merely the intensification of a previous chronic process, but is the primary affection.

A most detailed account of the etiology of suppurating kidney is given by Klebs (Handbuch der pathologische Anatomie). He is the first to alludes to the fact before stated that suppurative nephritis may occur, without any continuity of the inflammation with that in the bladder. This was taken by Beckmann to be a proof that the inflammation of the kidney is due to blood-poisoning. Klebs, however, states that he has discovered that it is due to the presence of minute organisms, similar to those found in decomposing urine, which find their way up from the bladder, multiply in the cortex, fill up the tubules, and then force their way into the insterstitial tissue, setting up inflammation terminating in suppuration, and he proposes to give the affection the name of Parasitic Nephritis.

That organisms are to be found in the pelvis and in the tubes of the pyramids in cases in which decomposition has extended to those parts, is no doubt true, but no other pathologist has at present confirmed the observation of Kleb in all its details. I have examined many specimens with Kleb's illustrations before me, and have failed to recognise either the organisms or the primary change in the epithelium as described by him. It must also be borne in mind that observers of the very highest ability have been deceived by the appearances produced by chromic acid, and have described granules due to this reagent as micrococci.

Dr. Dickinson, in a very able paper in the Medico-Chirurgical Society's Transactions, 1873, lays great stress upon the fact that the dissemination of the intertubular inflammation is chiefly in the course of the veins. He says, "This disorder has its origin in the regurgitation of urine charged with morbific products. This, forced backward by the retention general in these cases, distends or occupies the straight ducts. Thence by transudation, or similarly, it enters the neighbouring blood-vessels, and charges them with an infection resembling in its results that of pyæmia. This is distributed by the veins to the rest of the glands, sowing abscesses in their course." The theory is founded upon the almost constant association of suppurating kidney with decomposing urine, and upon the facts that clots are frequently found in the small veins of the kidney, and that the abscesses and lines of interstitial inflammation follow the course of the veins. On the other hand, as before stated, suppurative nephritis is not invariably associated with putrid urine in the pelvis of the kidney. Nor, so far as my observations go, are the clots in the vein by any means a constant occurrence. Although the chief lines of inflammation do follow the course of the veins, it must be remembered that in the same situation run the chief lymphatics, and there also the interstitial tissue is most abundant—Dr. Dickinson compares the condition to pyæmia, and suggests for the affection the name of the Uro-septic Kidney. He says, "the condition of the kidney may be described as one of pyæmia arising within itself. It has a close resemblance to that caused by a distant infection, differing from it in the usual dilatation of the urinary outlets, and in the fact that, while with pyæmia from a remote source the *materies morbi* is necessarily distributed by the arteries, in the condition under discussion it is scattered by the veins into which it was first received." There seems, however, to be one serious objection to this theory; the veins from the pyramids do not in any way communicate with the cortex. Those from the cortex, as well as those from the pyramids, empty themselves into the large veins at the cortico-medullary junction. If, therefore, thrombosis of the small veins of the pyramids, accompanied by subsequent softening of the clot, formed an important part of the process, the broken down clots would be carried on into the larger veins, and so into the general circulation and give rise to ordinary pyæmia. It seems therefore most probable that the coagula sometimes seen in the small renal veins are the result of the diffuse inflammation going on round about them and not the cause of the spread of the process.

If therefore the structure chiefly influenced by the presence of septic matter is not the epithelium and not the vessels, there are only two tissues upon which it can be reasonably supposed to act—the connective tissue and the lymphatics; and these it is impossible to separate in any way in such an organ as the kidney. The condition is one in which the lymph spaces and lymphatic vessels would be singularly liable to be affected. The pelvis of the kidney, and probably also the straight tubules for a greater or less distance, are filled with putrid urine at some degree of pressure. The contact of this irritating fluid damages the epithelium and causes its rapid desquamation. The septic matter then passes readily through into the intertubular lymph spaces of the kidney and excites a diffuse inflammation, which spreads rapidly towards the cortex between the tubules. As the connective tissue is most abundant in the course of the interfascicular veins, that is the line in which the process extends.

It has been suggested by Dr. Goodhart of Guy's Hospital, in the *Guy's Hospital Reports*, 1875, that in some cases the diffuse inflammation may be erysipelatous in character. He brings forward some interesting evidence in favour of the possibility of such an occurrence. But erysipelas could at most only account for a very small percentage of cases, and there are no means by which its presence can be definitely proved.

Dr. George Johnson has suggested that in some cases the localised abscesses may be due to rupture of the tubules, and from the pressure to which they are exposed, and escape of the urine into the intertubular tissue. Such an occurrence is of course possible, but there is no evidence to prove that it ever happens.

Lastly, suppurative nephritis was formerly supposed to be a simple catarrhal inflammation, extending from the bladder to the urinary tubules. The microscopic appearances in the kidney are sufficient to disprove this.

SYMPTOMS.—*Of the Chronic Interstitial Nephritis and Dilatation of the Kidney arising from increased Urinary Pressure.*—There are very few symptoms of any kind by which these conditions can be recognised during life, yet it is of great importance to the surgeon to ascertain, if possible, the exact state of the kidney before undertaking any serious operation. A sacculated kidney, although causing very little inconvenience to the patient under ordinary circumstances, is in a condition very prone to pass on into acute and fatal inflammation if exposed to the reflex irritation arising from an operation, or to the direct effect of putrid urine in the pelvis.

Secondary chronic interstitial nephritis causes no œdema, no marked anæmia, and no alteration in the function of the skin. There is no pain or tenderness in the renal region, and it is very rarely that palpation can give any information. The kidney might in some rare cases be felt to be enlarged, but it is very seldom that the dilatation proceeds to such an extent as to render this possible. In the earlier stages before much dilatation has occurred, nothing is recognisable. The examination of the urine is almost always rendered more or less difficult by the admixture of pus and mucus from the bladder, and occasionally by blood, arising in consequence of the primary disease. Under these circumstances it is sometimes possible to obtain urine of comparative purity in very small quantity by washing out the bladder with warm water, and then collecting through a catheter the first few drops that enter the bladder. The most marked change in the urine is its increase in quantity and its low specific gravity, indicating an increased blood-pressure in the Malpighian bodies, due to the obstruction of the venous circulation by the intertubular new growth. In order to ascertain this, twenty-four hours' urine must be collected, and the specific gravity of the whole taken. Great errors are likely to arise if this precaution be neglected.[1] The urine may be absolutely free from albumen. In a very typical case of simple dilatation of the kidney, arising from the pressure upon the ureters of two enormous sacculi, which projected from the bladder just behind the trigone, and reached on one side to the liver, and on the other to the spleen, the urine was found to be perfectly clear, free from albumen and casts, and of a specific gravity of 1009. Yet in this case the post-mortem examination showed the pyramids almost destroyed, and the cortex under the microscope exhibited a high degree of corpuscular infiltration of the intertublar tissue. There was not and never had been any cystitis, and beyond the sacculi no disease was found. The patient died suddenly during the aspiration of one of the sacculi for the purposes of diagnosis. In another case of prostatic disease lately under my care in University College, the patient, for some weeks before his death, passed from five to six pints of urine daily, of a specific gravity of 1003 to 1004, and containing merely a trace of albumen. The fatal termination of this case was due to an acute attack of interstitial nephritis. These cases clearly show that the absence of albumen is no evidence that the kidney is healthy, and if relied on may lead to most fatal errors in the choice of an operation. If any casts are present they are few in number, and hyaline in character. No other renal derivatives of any importance are found in the urine. I attempted in a few cases to ascertain the condition of the kidney by estimating the excretion of urea, but I found that the increased quantity of urine passed fully made up for the small proportion of urea in each analysis, and the daily quantity excreted was, as far as could be ascertained, normal—even in patients suffering from sub-acute attacks of interstitial nephritis. The quantity and specific gravity are then almost the only guides we have, and they are by no means certain. It is surprising how much dilatation, and how great an amount of destruction both of the pyramids and cortex, may occur without the excretion of urine being affected so much as to impair the health of the patient. This is probably due to the fact that the part destroyed performs merely the function of a duct, and that the epithelium of the true secreting part of the kidney is but little altered.

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[1] In a case of lithotrity, under my care at University College Hospital, the specific gravity of the first sample of urine examined was 1003, but that of the whole twenty-four hours was 1018.

Symptoms of Sub-acute Diffuse Interstitial Nephritis.—Sub-acute interstitial nephritis may last for weeks, or in some cases for months. It runs an irregular course, marked by occasional exacerbations and intervals of apparent recovery. The periods of improvement usually coincide with some diminution of the local irritation at the primary seat of disease. In a very typical case, secondary to disease of the prostate, with cystitis and decomposition of the urine, under my care in University College Hospital, the patient was twice admitted and sent out again, relieved of all urgent symptoms, the improvement being brought about each time by washing out the bladder with an antiseptic solution, and thus diminishing the cystitis consequent upon the ammoniacal urine. The urine passed immediately after the bladder was washed out was always acid, thus showing that the decomposition had not extended up the ureter to the kidney. This case ultimately terminated fatally from an acute attack with suppuration in the kidney. In sub-acute interstitial nephritis, if the attack arises as the result of some surgical operation, the commencement is usually marked by a rigor. In other cases, chills frequently occur at the beginning of the attack, and occasionally during its progress, but a distinct rigor is rare. The general health suffers considerably; the patient is weak and languid, and slowly but steadily emaciates. The tongue is covered with a dirty, whitish fur, which has a great tendency to dry. The mouth is clammy, and there is frequently considerable thirst. The appetite is bad, and there is occasionally a tendency to nausea, but actual vomiting is rare. Occasionally there is diarrhœa, but by no means constantly. Examination of the kidney by palpation gives but little information. It is seldom sufficiently enlarged to be clearly felt through the abdominal walls, and there is usually no tenderness on deep pressure. There is, in fact, very little pain of any kind, and unless the patient is disturbed by some intense suffering from the primary disease, he is usually in a very placid state, almost comfortable—often drowsy, like a man slightly under the influence of opium. There is no dryness of the skin; in fact it is usually moist and clammy. The pulse presents nothing characteristic. The temperature is peculiar and very characteristic. For weeks together it may be normal or below normal, in the morning, and ranging from 99° to 101° in the evening. As an example the following may be given, taken from a case of lithotomy nearly six weeks after the operation:—

Morning.	Evening.	Morning.	Evening.
97·4° Fahr.	99·1° Fahr.	98·3° Fahr.	100·1° Fahr.
98·3	98·9	98·2	100·0
97·0	100·5	—	100·8
98·0	101·1	98·5	—
98·6	100·1	98·4	100·2
98·4	101·2	98·2	99·0
97·0	101·2	97·4	100·6

This patient was discharged from the hospital without any improvement of the symptoms, and his ultimate fate could not be ascertained. An occasional observation of the temperature is valueless. In order to draw a useful conclusion, it is necessary to use the thermometer morning and evening for a few days. I have not unfrequently seen cases in which the temperature has been supposed to be normal, from the observations being made only in the morning. The evening rise is seldom great, 101° or 102° being usually the maximum. If the morning temperature rises also above normal, and the evening reaches a higher point, it is to be feared that the sub-acute inflammation is passing on into the acute form. The urine is in most cases abundant, and of low specific gravity, especially in those cases in which obstruction to the flow of the urine has existed for a long time. Cases, however, do occur in which there is little or no alteration either in the quantity or specific gravity. The amount of albumen is not great, in most uncomplicated cases it is merely a trace; but usually the estimation of the exact amount is impossible from the admixture with the urine of pus, mucus, and blood from the bladder. Microscopic examination is also difficult for the same reason. In some cases, however, it is possible to find some few hyaline casts, and a few renal-epithelium cells; but usually they are held in suspension in the thick, ropy mucus, so that their discovery is merely a matter of chance. Occasionally casts are found with leucocytes imbedded in them. These indicate that the sub-acute condition is verging on the acute.

The symptoms above described may last for a long time, the patient gradually becoming weaker and weaker, until he is carried off by some intercurrent disease or dies of pure exhaustion. Much more frequently the fatal termination is due to an acute attack, usually accompanied by suppuration of the kidney, excited either by the irritation of some surgical operation, or by the extension of decomposition from the bladder upwards into the ureter and pelvis of the kidney. On the other hand, if the primary disease be relieved, and all sources of irritation removed, the symptoms may gradually subside, and the patient return apparently to perfect health.

It is often difficult when the patient is suffering much from the primary affection to ascertain with certainty how much of the constitutional disturbance is due merely to this, and how much to secondary renal mischief, especially in cases of stricture complicated with perinæal abscesses. However, if we find a dry tongue, loss of appetite, a nocturnal elevation of temperature, and a history of progressive weakness and emaciation, and especially if with these the urine is abundant and of low specific gravity, a condition of sub-acute interstitial nephritis must be suspected, which by any injudicious surgical operation, undertaken for the relief of the primary disease, might easily be intensified to such a degree as to become incompatible with life.

Symptoms of Acute Interstitial Nephritis, or of Suppurative Nephritis.—There are no definite symptoms by which acute diffuse interstitial nephritis can without suppuration be distinguished from suppurative inflammation of the kidney; but whenever the symptoms proceed rapidly to a fatal termination, suppuration may be suspected. The symptoms of acute inflammation often supervene on those of the sub-acute form just described. The immediate cause of the attack is frequently some surgical operation, such as the passage of a catheter, lithotomy, or lithotrity. An operation upon the deeper parts of the urethra is a frequent cause, but one on the anterior part, in front of the scrotum, is rarely, if ever, followed by any secondary disturbance. In other cases the attack is induced by extension of the decomposition from the bladder into the ureters and pelvis, which may be recognised by the impossibility of any longer obtaining acid urine from the bladder, even after most carefully washing out its cavity with an antiseptic. The attack commences with a violent rigor, during which the temperature rises to 105° F. to 106° F. It is followed by profuse sweating. After the rigor the temperature falls, but still remains a little above normal; and there is a regular evening rise of one or two degrees. Several rigors may occur, or only one. The tongue becomes at first furred, but very soon dry, red, and fissured, looking like a piece of broiled ham, and sordes accumulate on the teeth and lips. The loss of appetite is complete, and there is almost invariably more or less nausea: actual vomiting is not of very frequent occurrence. Diarrhœa may occur, but is not usually a marked symptom. Emaciation occurs with great rapidity, a few days sufficing to make a great alteration in the patient's appearance. The skin is usually moist throughout, except during the rigors. Most frequently it is covered by a clammy perspiration. The mental condition of the patient is very peculiar. He is often in a happy, dreamy state, closely resembling the effects of opium. There is seldom any delirium, and convulsions are of great rarity. The pupils are in some cases contracted, in others of normal size. As the case advances there may be muttering delirium, the pulse becomes weaker, the patient becomes gradually insensible, and dies. There is never the profound coma so often found in death from acute Bright's disease. Before death the temperature falls, and may often remain normal for the last few days of life. Occasionally the patient complains of more or less pain in the lumbar region, and there is almost invariably tenderness on deep pressure over the kidney. The urine in these cases is usually in so foul a state that accurate observation of it is impossible. It is frequently tinged with blood, and in some rare cases it is greatly diminished in quantity, or even suppressed. Much more commonly it remains abundant to the end, and contains a fair quantity of urea. In one case in which I estimated the amount of urea, I found that over 300 grains were passed on the last day of life. The amount of albumen varies much, but it is usually moderate in amount, unless blood or pus is present in large quantities. The microscope shows the presence of renal epithelium in considerable abundance, with bladder epithelium, pus, and blood corpuscles. Occasionally, casts may be found. These are usually hyaline, but may be granular. In some rare cases pus casts may be observed.

It will be seen that, although the patient dies directly from the acute inflammation of the kidney, the symptoms do not resemble in any respect those of acute Bright's disease. There are no convulsions, no coma, no œdema, no dryness of the skin; and the urine, instead of being scanty and dark-red in colour, is usually abundant and only faintly tinged with blood. On the other hand the symptoms closely resemble, and in fact are often indistinguishable from, those of septicæmia. There is the preliminary rigor, the brown dry tongue, the rapid emaciation, the temperature high at first gradually falling below normal, the occasional diarrhœa, the drowsy condition, and gradually increasing insensibility without coma before death. In some cases also the skin may assume a yellowish tint. At the same time, the secretion of urine continues in fair quantity, and the patient cannot therefore be said properly to die of uræmia. It seems most probable that the death is in reality due to septicæmia, the septic matter being absorbed by the veins and lymphatics of the kidney in sufficient amount to cause death, while at the same time the power of elimination of the poison is more or less limited by the state of the kidney.

In some exceptional cases diarrhœa forms a very prominent symptom. In a young man, aged thirty, under treatment for stricture in University College Hospital, the most marked feature was profuse diarrhœa exactly resembling that of typhoid fever. The temperature, however, remained constantly below normal for ten days before death. The post-mortem examination showed the mucous membrane of the intestine, from the stomach onwards, in a state of intense congestion. The lower part of the ileum was extensively ulcerated. The ulcers were not confined to Peyer's patches, many were transverse in direction, and the edges were not undermined. Both kidneys were suppurating, but there was scarcely a trace of pyelitis.

Another variety of the disease is described by Malherbe in his admirable treatise on uræmic fever, in which abscesses, containing pus of a urinous odour, are found in the intermuscular connective tissue, in the subcutaneous tissue, and various other parts. It has not fallen to my lot to see such a case, and it seems doubtful whether the condition is not in reality a form of pyæmia.

The duration of acute interstitial nephritis is seldom more than two weeks, and it is very frequently fatal in a shorter time. Recovery not unfrequently takes place even after symptoms of a very severe character have set in. In a case of this kind which I had the opportunity of observing, in which lithotrity was followed by marked symptoms of acute interstitial nephritis, the patient suffered for two years from occasional attacks of vomiting, with drowsiness and pain in the back preceded by rigors. I never had the opportunity of examining the urine during these attacks, but they were apparently due to acute congestion of the kidney. They were always attributable directly to exposure to cold. The intervals between the attacks became longer and longer, and finally they ceased altogether, and for many years the patient has been in excellent health.

It will be seen that the above description of the symptoms of acute and sub-acute nephritis includes all those spoken of by various authors as belonging to urethral or uræmic fever. It is not the place here to discuss the dozen or more theories which have been invented to explain this febrile disturbance, which so commonly follows all operations involving the bladder, prostate, or bulbous portion of the urethra. All those which attribute the disturbance to the passage of the urine over a raw surface, absorption of urine from the wounded part, or absorption of septic matter, are rendered improbable by the fact that operations in front of the bulb very rarely if ever give rise to rigors and fever. The most probable explanation of the symptoms is that the irritation of the nerves of the bladder or urethra caused by the operation is reflected to the kidney. As the nerves of these parts are chiefly from the sympathetic system the stimulation is followed by a contraction of the vessels of the kidney, giving way after a time to dilatation, slowly increasing till the kidney is in a state of active congestion. As the dilatation of the renal vessels takes place there is a corresponding contraction of the vessels of the skin, and hence the sensation of cold culminating in a rigor, in part also the elevation of temperature. The intimate relation between the skin and the kidneys is probably maintained by the vaso-motor nervous mechanism. Contraction of the vessels of the skin is accompanied by a dilatation of the vessels of the kidney, and there is therefore nothing unreasonable in supposing the converse, that a dilatation of the renal arteries may cause a contraction of the vessels of the skin. Moreover, direct evidence was adduced in a former part of this article to show that there is good reason to believe that mechanical violence to the urethra or bladder causes dilatation of the renal vessels and acute hyperæmia of the kidney. If the kidney be healthy the congestion soon relieves itself by an increase in the flow of urine, and no evil consequences result; but if it be already suffering from chronic interstitial nephritis, with more or less venous obstruction, such engorgement of the vessels may take place as to lead to complete blocking and stasis, accompanied either by suppression of urine or the excretion of a very small quantity darkly tinged with blood. In other cases the attack of acute hyperæmia may aggravate the existing inflammation, and the sub-acute nephritis may rapidly become acute.

Suppression of urine after operations upon the urinary organs is of rare occurrence; and, although many cases have been recorded, there are few in which a microscopical examination of the kidney has been made after death. It is probable that in all cases the kidney is extensively diseased before the operation. A case lately occurred in University College Hospital in which I had the opportunity of examining the kidneys after death. In this case perinæal section was performed for a very old and intractable stricture. Eleven hours after the patient had a violent rigor; the temperature having risen from 99·6° F. to 102° F. since the operation. In the first twelve hours about half a pint of urine escaped into the bed, and from that time till his death on the third day no urine was passed. At the post-mortem examination the bladder was found to be empty. The kidneys were large, tough, and pale in colour, having the ordinary appearance of sub-acute interstitial inflammation. Microscopic examination showed abundant small round-cell infiltration of the inter-tubular tissue throughout the kidney. The vessels were not engorged with blood. The epithelium was a little swollen and granular, but the nuclei of the cells stood out clearly, and the lumen of the tubules was not choked. It was evident that the cause of the suppression was not the obstruction of the tubules by altered epithelium, and must therefore have been due to some alteration in the supply of blood. It is impossible to conceive that it was due to a prolonged spasm of the arteries lasting for three days, or to such a general lowering of the blood-pressure that fluid ceased to flow through the walls of the Malpighian vessels. The absence of vascular engorgement after death would negative the supposition that it was due to choking of the renal vessels during an attack of acute congestion, and the only conclusion that can be arrived at is that during the first few hours the renal vessels became dilated, and that in consequence of this so great an inter-tubular exudation took place as to compress the small arteries to such an extent as greatly to lower the blood-pressure in the Malpighian tufts. At the same time the tubules would be also compressed, so that if any fluid passed into them from the Malpighian tufts its passage downwards would be obstructed. Thus by lowering the blood-pressure in the vessels and increasing the urinary-pressure in the tubules, the two soon become equal, and all flow from the Malpighian tufts must have ceased. With such an amount of disturbance in the vascular supply it is not possible that the epithelium could continue to perform its excretory function, and thus complete suppression of urine would result.

In other cases, however, far from being anæmic, the kidney has been found to be intensely congested, and the tubules to contain extravasated blood. In this form the suppression of urine would be due to such an amount of engorgement of the vessels of a kidney already diseased as to lead to complete stasis.

Diagnosis.—The diagnosis of the effects of simple pressure has already been sufficiently alluded to. As it gives rise to few marked symptoms, it is not likely to be mistaken for any other disease, the only danger is that it should be overlooked.

The sub-acute form of interstitial nephritis may be mistaken for the effects of the irritation, pain and want of rest, caused by the primary disease; and, possibly, for chronic blood poisoning from absorption of septic matter from the bladder. These errors can only be avoided by carefully examining the urine, if it can be collected, as to quantity and specific gravity, albumen and casts, and by the observation of the morning and evening temperature for a few days. If the quantity is large, the specific gravity low, the albumen small in amount; if a few hyaline casts, and possibly renal epithelium, are present; and if the temperature shows a regular and marked evening rise, while in the mornings it is normal or nearly so, there will be strong reasons to believe that sub-acute interstitial nephritis exists. The progressive emaciation, feeble appetite, thirst, dry tongue, tendency to nausea, and occasional chills are also valuable diagnostic signs. Acute interstitial nephritis, with or without suppuration, may be mistaken most commonly for pyæmia or septicæmia. It may in some cases resemble septic peritonitis or typhoid fever. From *pyæmia* it may be distinguished by the absence of secondary abscesses in the ordinary parts in which they occur in this disease. The temperature also usually falls below normal, or nearly to normal, some days before death, while in pyæmia it remains high till death. The rigor may be single or multiple in both diseases. From *septicæmia* the diagnosis is often impossible. In fact, as before stated, in many cases the real cause of death is not the damage to the kidney, for the excretion of urine remains abundant to the last, but absorption of septic matter from the abscesses or from the putrid urine in the pelvis. The drowsy state of the patient, the tenderness and pain in the loins, the comparatively early fall in the temperature, and the vomiting, may serve to point to the nature of the case, but the diagnosis must in most cases be somewhat uncertain. Septicæmia is not, however, a common occurrence except as a result of septic inflammation of the kidney, and errors in diagnosis cannot frequently occur. It is only in very rare and anomalous cases, complicated with much diarrhœa, that the symptoms resemble those of *typhoid fever;* and the diagnosis is readily made by the temperature, which usually falls to normal some days before death, and is seldom so high as that of typhoid fever. The absence of spots, the tendency to vomiting, the early period at which the tongue becomes dry and brown, also serve as valuable distinctive features. Occasionally the disease closely resembles that form of *peritonitis* which arises from the presence of septic matter in the peritoneum, that is to say, in which the symptoms of septicæmia caused by absorption of the infective products mask the ordinary signs of inflammation of the peritoneum. Such peritonitis may arise from perforation of the bladder. In such cases there is no rigor, the pain is usually greater, and the vomiting is more severe than in acute suppurative nephritis.

Patients who have at some time suffered from *ague* are very liable to the occurrence of rigors after operation on the urinary organs. In such cases there is therefore additional difficulty in making a diagnosis.

PROGNOSIS.—In the chronic forms of secondary renal disease the prognosis depends much more on the primary than on the secondary affection. If the original disease is curable, the patient may apparently completely recover. In reality, however, the structure of the kidney is always somewhat damaged, and it is left in a condition resembling the ordinary granular contracted kidney. In this state, exposure to cold or other causes of renal congestion may light up fresh disease.

In the sub-acute forms, if the primary cause be removable the prognosis is not bad, but there is always the danger that the necessary operation may light up acute inflammation which may rapidly prove fatal. If the primary cause be not removable, the case will certainly terminate fatally before very long. Most likely after various exacerbations and partial recoveries, an acute attack will end in suppuration of the kidney and death. The acute forms are always very dangerous, drowsiness, vomiting, a very dry tongue, much tenderness in the loins, and an abnormally low temperature are all very grave signs—if the urine is very foul, and the bladder cannot be cleansed by washing it out, the prognosis is very bad. Suppression of urine, if lasting over one day, is almost always fatal.

In the so-called urethral fever, in which the whole affection consists merely of acute congestion of the kidney following an operation on the urinary organs, there is little danger if the temperature returns to normal soon after the hot stage of the rigor. The longer the symptoms last the more reason there is to fear that there is serious structural change in the kidney in addition to the temporary hyperæmia.

TREATMENT.—The treatment of the acute and even of the sub-acute varieties of interstitial nephritis is so unsatisfactory that every effort should be made to prevent their occurrence. The three great causes of secondary renal mischief should therefore be constantly borne in mind—increased urinary pressure from obstruction to the flow of urine, reflex irritation from the bladder or urethra, and decomposition of urine extending to the ureter and pelvis of the kidney. Almost without exception the causes of urinary obstruction require operative interference for their relief, and consequently, in order to remove the first cause of disease, the second must necessarily be more or less called into action; and as there is every reason to believe that the causes of decomposition of the urine come from without, and are in fact organisms which find their way to the bladder, and set up fermentative changes, the third may also be brought into play at the same time. The amount of irritation caused by the different operations on the urethra or bladder varies greatly. Thus the simple passage of a bougie causes less disturbance than the forcible dilatation of a stricture, or than internal urethrotomy; and the use of soft instruments which can scarcely injure the walls of the urethra less than the passage of metallic catheters. The surgeon, therefore, whenever he has a choice, should employ that mode of treatment which involves least mechanical violence to the part operated on. The operation should also be of as short duration as possible. In cases of stone in the bladder, the relief given by lithotomy is so complete, that it is often safer, when the kidneys are known to be diseased, to risk the single violent irritation of lithotomy than to expose the patient to the repeated and prolonged disturbance caused by lithotrity. Decomposition of the urine should if possible be prevented, and as there is no doubt that the causes of decomposition are frequently introduced on the catheter, all instruments should be kept scrupulously clean, and the oil used should contain one-twelfth part of carbolic acid. Decomposition however does occur in cases in which no instrument has been used, but only when cystitis is present. This is not difficult to explain on the germ theory of decomposition. In health the urethra is washed out thoroughly at each time that urine is passed, and in the intervals is a closed tube just moistened by a trace of healthy mucus; but in cystitis the ropy tenacious mucus, coming away at the end of micturition, leaves a layer adherent to the walls of the urethra, which is not washed cleanly out by the subsequent acts of micturition, and in which at the temperature of the body organisms will develop with the greatest rapidity; and by this means they find an introduction to the bladder. It is very difficult therefore, when there is much cystitis, to prevent ammoniacal fermentation taking place. If then it cannot be prevented, it must be limited as far as possible by washing out the bladder at least twice a day with some antiseptic solution. A very useful solution for this purpose is sulphate of quinine, g. ij., and dilute sulphuric acid, m. ij., to one ounce of water. Carbolic acid is usually too irritating, as, in order to be effectual, it must be used in the proportion of one part to fifty of water. Condy's fluid is often very useful, but its effects are not so good as those of quinine.

It is most important that no operation, unless it be one of emergency, should be undertaken while the patient is suffering from sub-acute interstitial nephritis. If possible, in every case of urethral or vesical disease, the urine should be collected for the twenty-four hours and carefully examined as to quantity, specific gravity, albumen, &c. This should be repeated for two or three days, and at the same time the morning and evening temperature should be observed. If any reason is found to suspect renal mischief, the patient should be confined to bed, the bowels should be well opened, and the skin should be made to act by hot baths or vapour-baths. A mustard plaster might also be applied over the loins. He should be kept on moderate diet, and stimulants as far as possible excluded. No drug could be of any special value. If the urine is foul the bladder should be washed out regularly, if the catheter can be passed easily, as the passage of the instrument is less likely to do harm than the presence of decomposing urine in the bladder. Under this treatment the symptoms will frequently, in a few days or a week, so far subside as to render the operation safe. After the operation in such cases the patient should be wrapped up warmly in bed, to promote as far as possible the action of the skin. Quinine, opium, eucalyptus globulus, and many other drugs have been recommended to prevent the occurrence of the so-called "urethral fever" which often follows operations on such cases. I cannot say that I have ever seen the least benefit from any, except perhaps from opium, which, by increasing the action of the skin, tends to relieve the congestion of the kidney.

When the symptoms of acute interstitial nephritis are well marked, treatment is often of but little use. Hot baths, wet packing, or vapour baths should be employed to promote the action of the skin; dry cupping, or if the patient be very strong, wet cupping over the kidneys, followed by hot fomentations, should be tried. The bowels should be gently acted on by purgatives, unless there is already diarrhœa. Opium may in many cases be given without danger; and, if the patient is suffering pain, should always be employed. Quinine has been given as in septicæmia, but its use has not proved of any special service. No operation beyond the passage of a catheter when necessary, either to draw off the urine or wash out the bladder, should ever be undertaken. Heroic operations performed during acute interstitial nephritis, with a view of relieving local irritation, are invariably fatal.

CALCULOUS DISEASE OF THE KIDNEY.

By W. R. Basham, M.D., F.R.C.P.,

AND

Frederic T. Roberts, M.D., F.R.C.P.[1]

Definition.—A renal calculus or "stone" is a concretion formed in some part of the kidney, varying in its size and other characters, as well as in its composition, but larger than the minute formations which constitute what is generally known as "gravel." A distinction has been made by French writers between "gravel stones" and "true renal stones," the former being of such a size that they are able to pass through the urinary passages; but no such artificial line of demarcation is of any practical advantage. A calculus, if it does not escape by the ureter, tends to set up various morbid conditions, the most important being congestion or inflammation of the substance of the kidney—*calculous nephritis;* inflammation of its infundibulum and pelvis—*calculous pyelitis;* or a combination of these lesions—*pyelo-nephritis.* Ultimately the renal structure may be entirely destroyed. The symptoms may merely indicate functional disturbance of the kidneys, or local mechanical irritation; or they arise from the passage of the calculus along the ureter; or they may be the result of the lesions which it originates; and they vary accordingly. In exceptional instances the stone becomes so encysted and encapsuled as to prove harmless, and to give no clinical sign of its presence.

Synonyms.—Renal Calculus; Stone in the Kidney; Nephrolithiasis; Calculi Renum.

Pathology and Ætiology.—By far the larger number of urinary calculi are formed originally in connexion with the kidney (according to Heller the proportion is 100 to 1), and those which are found in the bladder have generally passed along the ureter from the renal pelvis to this viscus. Their formation may commence either in the uriniferous tubules, where they may remain permanently, or in the calices, infundibulum, or pelvis of the kidney. When the concretion starts in the substance of the kidney, it is usually, when still minute in size, conveyed by the urine into the pelvis of this organ, where it subsequently becomes enlarged by a continued deposit upon its surface.

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[1] This article was left in an imperfect and unfinished state by the late Dr. Basham, and has been revised and completed by Dr. Frederic T. Roberts.—Editor.

The precise pathology of the formation of renal calculi is still not satisfactorily settled, but no doubt it differs in different cases, and the proximate cause may probably be referred to one or more of the following conditions:—1. The urine may contain an excess of one of its normal constituents, a portion of which is under favourable circumstances deposited, so as to originate a concretion. Thus there may be an undue proportion of uric acid, urates, or oxalate of lime, which leads to the precipitation of either of these substances, and the production of corresponding calculi. 2. The proportion of the ordinary urinary ingredients which are liable to form calculi being within the normal limits, the conditions of the urine may be such that it cannot retain one or other of these in solution. This may depend on its reaction. Thus highly acid urine is an imperfect solvent of uric acid and urates, which may accordingly be precipitated. This undue acidity has been attributed to the presence of an excess of acid bi-phosphate of soda, withdrawing soda from the urate; or to acid fermentation in the urine, with decomposition of its colouring and extractive matters. In rare instances also urine which is alkaline from the presence of fixed alkalies deposits basic phosphate of lime or carbonate of lime; while an alkaline condition of this excretion, due to its decomposition and the consequent production of carbonate of ammonia, is very liable to lead to the precipitation of other varieties of phosphate of lime, as well as of ammonio-magnesian phosphate, mixed with a small quantity of urates and carbonate of lime, and although this usually happens in the bladder, it occasionally occurs in connexion with the kidney. Again it has been affirmed by Heller that a deficient amount of chloride of sodium or of alkaline phosphates in the urine diminishes its solvent power over uric acid and urates, and thus favours their precipitation. A low temperature of the urine has been supposed to have some influence in favouring the development of calculi in old people. 3. In very exceptional cases the urine contains some abnormal constituent, which is but slightly soluble, and therefore is readily deposited. The most important substance which may thus form a calculus is cystine, of which in extremely rare instances renal calculi are found to consist. Examples are recorded by Dr. Church,[1] where the calculus was covered with a thick layer of viscid muco-purulent material, in which were numerous plates of cholesterine; and by Dr. Leared.[2] Still more rarely xanthine is present in the urine, and forms a calculus. 4. An important element in the origination of, at any rate, a large number of renal calculi is the presence of some material, either forming a nucleus upon which the one or other of the urinary constituents is deposited; or with which they combine from the very outset; this combination determining their precipitation. Indeed, it has come to be a question whether the primary formation of a calculus is ever due to mere precipitation, or whether there is not always a

[1] Pathological Transactions, vol. xx. p. 240.

[2] Ibid. vol. xxiii. p. 165.

basis of animal matter which causes the first separation of the urinary ingredient by which the nucleus of the stone is originated. Occasionally a distinct nucleus of coagulated blood, or of inspissated mucus or pus, is found in the centre of a calculus, to which this evidently owes its development. Dr. Curnow[1] has described calculi of oxalate of lime, each having a large nucleus of blood-clot, and which he therefore considered were sequential to one or more attacks of renal hæmaturia. In rare instances entire concretions seem to be formed of coagulated and decolorized blood. Casts in the renal tubules have been credited with the origin of calculi, forming points for the deposit of crystals, especially those of oxalate of lime. Greisinger also found the eggs of the *Bilharzia Hæmatobia* forming the nucleus of a stone in Egypt, and these have been supposed to account for the frequency of calculous disease in that country. Dr. William Roberts suggests that the little clumps of urate of soda which are not uncommonly deposited in the urinary passages in connexion with the febrile complaints of infants and young children, may be retained in the pelvis of the kidney, and become the nucleus of future calculi. With reference to the influence exercised by a basis of animal matter in laying the earliest foundation for the development of renal calculi, important observations have been made, especially by Dr. Vandyke Carter,[2] and Dr. Ord,[3] founded on the structure of these concretions, as revealed by microscopic examination. Mr. Rainey showed that in the shells of animals the mineral salts are precipitated in certain forms incorporated with animal matter, and he produced similar deposits artificially, by mixing saline solutions with organic substances, the process being termed by him "molecular coalescence." Dr. Carter has endeavoured to explain the production of renal calculi in a similar manner, having found the forms of mineral deposit to coincide with those observed by Rainey. The researches of this observer, as well as those made by Ord, have revealed the fact that globular forms, and not ordinary crystals, are produced when mineral salts are slowly precipitated in a colloid medium. In his investigations Carter found that the nucleus of a calculus almost always consists of these globular forms of urates or oxalate of lime, and that it also contains a colloid matrix. Hence the theory that many calculi at any rate originate in the presence of a little mucus or other colloid substance in some part of the renal apparatus, with which urates or oxalate of lime become mixed, and these are then precipitated in a globular form.

A calculus having once been started, it increases in size by a continued deposit upon its surface, this usually assuming a stratified arrangement. The layers may consist of a similar material, so that the stone is uniform in its composition, though the nucleus is often different from the subsequent deposit; or two or more substances may alternate more or less regularly. In the great majority of instances the deposits are *primary*, that is, they are composed of ingredients separated from the urine as it is first excreted; it may happen, however, that a calculus having remained in the renal pelvis, and having set up pyelitis, while at the same time the urine is retained in this part, the products of inflammation cause this fluid to decompose and to become ammoniacal, under which circumstances, as already pointed out, calcic and ammonio-magnesic phosphates are precipitated, which form what are termed *secondary* deposits. The size to which a stone may thus attain is very considerable, and it may ultimately cause complete destruction and obliteration of the renal apparatus.

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[1] Pathological Transactions, vol. xxiv. p. 148.

[2] On the Microscopic Structure of Urinary Calculi. London: 1873.

[3] Medico-Chirurgical Transactions, 1875, and Quarterly Journal of Microscopic Science, vol. xii. new series, p. 219.

[4] On the Mode of Formation of Shells, &c. London: 1858.

Having thus far discussed the conditions under which renal calculi are formed, it remains to be considered what causes originate, or predispose to these conditions.

1. *Hereditary Influence.*—The tendency to the development of calculi seems to be in many instances hereditary, and this especially applies to certain varieties, particularly the cystine-calculus, though it is not always possible to trace family predisposition even in this form, as in the case already mentioned which is recorded by Dr. Leared. The hereditary tendency is presumed to depend upon a peculiar diathesis or constitutional condition, and in past times each variety of calculus was attributed to a special diathesis—the uric or lithic acid diathesis, the oxalic acid diathesis, &c. That a condition of system favourable to the excessive formation of these products may be transmitted cannot be doubted, and it probably accounts for a certain proportion of cases of renal stone. This is one of the pathological consequences of the gouty constitution.

2. *Age.*—Calculus is of much greater frequency during childhood than at other periods of life. It may be found even in the fœtus. The disease is most common under five years of age; next from this period until about fifteen; after this its frequency diminishes until middle life, but subsequently as age advances it again becomes more prevalent until about sixty-five, when the proportion of cases becomes once more lessened.

3. *Sex.*—Males are much more liable to calculus than females, and the proportion of deaths in the former is also much greater. This statement applies both to children and to adults.

4. *Habits of Life.*—There can be no doubt but that excessive eating, especially of meat and other highly-nitrogenized food, over-indulgence in alcoholic drinks, want of exercise, and an indolent and sluggish mode of living, with other habits which tend to develop the gouty condition, and to lead to the formation of excess of urates and oxalates, materially assist in the causation of renal calculus in some cases. This accounts for the comparative frequency of uric acid calculus among the better classes after middle life. At the same time too much stress must not be laid on these conditions, as the complaint frequently occurs under totally opposite circumstances, and it must be borne in

mind that a stone may be formed when there is no excess of its constituents in the urine. Oxalate of lime calculus has been attributed to the excessive consumption of vegetables containing an abundance of oxalates, especially rhubarb and sorrel.

5. *Climate and Locality.*—It is found that renal calculus occurs in every variety of climate. Formerly it was believed to be chiefly met with in damp and cold climates, and that it did not occur in tropical regions. The complaint has, however, been found to exist largely in India and other hot climates, and therefore such a notion is proved to be incorrect, no region being exempt from the disease. At the same time it does prevail more in certain countries and districts. The disease is common in England, especially in the eastern counties of Norfolk and Suffolk, and next in the north-midland counties. Dr. William Roberts[1] gives the following as the proportion of deaths (in males) from stone in each of the eleven registration districts of England and Wales, for every 100,000 deaths from all causes, in the ten years, 1857-1866:—North-Western 34; South-Western 46; Northern 54; West-Midland 64; South-Midland 71; Yorkshire 75; London 90; Monmouthshire and Wales 91; South-Eastern 93; North-Midland 98; Eastern 115. Stone is also prevalent in France, Iceland, Egypt, and the Netherlands, though less so in the last-mentioned than formerly. The complaint is rare in Sweden and Norway. The endemic occurrence of calculus in some districts has been attributed to the use of drinking-water containing an undue proportion of lime-salts, and the influence of such water in originating stone has been extensively believed in; but there seems to be no adequate foundation for such a belief.

6. *Renal Disease.*—Previous disease in connexion with the kidney may have considerable influence in originating a calculus. For instance, such a disease may cause hæmorrhage, and thus give rise to a clot forming a nucleus; or there may be catarrh of the uriniferous ducts, or more or less pyelitis, leading to the production of mucus or pus, which determines the precipitation of the constituents of the urine, thus forming the basis of a stone.

ANATOMICAL CHARACTERS.—This part of the subject may be discussed under the following heads:—

1. The general description of a renal calculus.
2. The description of its several varieties.
3. The direct effects which stone is liable to produce on the renal apparatus or neighbouring structures.
4. Its remote effects and complications.

1. *General Description.*—A renal calculus is usually more or less oval or circular in shape, but it may be irregular or branched if it is long retained, becoming moulded to the interior of the renal outlet. Much variety is presented as to size, number, condition of the surface, colour, degree of hardness, and other characters, these depending to a great extent on the nature of the concretions. On section it is

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[1] Urinary and Renal Diseases, third edition, 1876, pp. 272, 273.

generally seen to consist of a *nucleus* in the centre, outside which is the *body*, with, in exceptional instances, a still more superficial deposit, made up of secondary phosphates, and termed the *crust*. As a rule a concentric stratified arrangement is evident, the strata varying much in number and thickness, the one bearing an inverse proportion to the other, while they may present similar or different characters, and regularly alternating layers of different composition may be observed. Sometimes the layers are slightly wavy. Occasionally the structure is in the form of lines radiating from the centre, or the concentric and radiating arrangements may be more or less combined. In some calculi no regular structure is discernible. The *chemical composition* varies according to the nature of the stone, and when the strata are made up of different constituents, this is of course revealed by the chemical analysis. The nucleus may or may not be of the same composition as the remainder of the calculus. There is always a certain proportion of organic matter mingled with the essential constituent or constituents of which the several varieties of renal stone are composed.

2. *Varieties of Renal Calculi.*—The following are the varieties of renal stone met with, named according to their composition, the more prominent characters of which it will now be desirable to describe:—(*a*) *Uric Acid;* (*b*) *Urates,* especially urate of ammonia; (*c*) *Oxalate of Lime*—the so-called *Mulberry Calculus;* (*d*) *Phosphates;* (*e*) *Carbonate of Lime;* (*f*) *Cystine;* (*g*) *Xanthine;* (*h*) *Fibrin or Blood;* (*i*) *Mixed.*

(*a*) *Uric Acid.*—By far the largest proportion of renal calculi consists of this substance, of which it has been calculated that five-sixths of these concretions are made up. This is explained by its being such a very sparingly soluble substance; and although it may be met with at all ages, this variety of calculus is especially common after middle life, and is frequently associated with the gouty condition. Uric acid calculi are often very numerous. They vary much in size and weight, ranging from minute concretions, which border upon gravel, to a mass completely filling the renal pelvis. Usually they weigh from a drachm to an ounce, but may be much heavier than this. They are hard and dense, their specific gravity being about 1·5. In shape they are generally oval and elongated, or rounded, and more or less flattened. The surface is minutely tuberculated or mammilated. The colour varies according to the admixture of pigments: thus it may be light fawn, brick-red, or vermilion, or in exceptional instances reddish-brown or greenish. The broken surface presents usually a crystalline appearance in the smaller concretions, an amorphous one in the larger, which also have a peculiar wooden aspect. A concentric arrangement is evident, and the layers may be of different colours. Chemically uric acid calculi are characterized by being soluble in a very weak solution of carbonate of potash or soda, but insoluble in water, strong solutions of the alkaline carbonates, or dilute mineral acids. Uric acid is readily recognized by the *murexid test.*

(*b*) *Urates.*—These consist mainly, but not entirely, of urate of ammonia. Calculi made up wholly of urates are rare, and are almost limited to young children; but the nucleus of other calculi is frequently thus constituted. This variety of stone is in the form of soft, irregular concretions, which are characterized chemically by being soluble in hot water.

(*c*) *Oxalate of Lime—Mulberry Calculus.*—This substance rarely forms the whole of a stone, but generally alternates with layers of urates. Calculi of oxalate of lime are never so numerous as those of uric acid, and there may be but one, or several may be present. In the case already mentioned, which is recorded in the Pathological Transactions by Dr. Curnow, there were several calculi in both kidneys, consisting externally of pure oxalate of lime, with a large nucleus of blood-clot. In size they vary considerably, from not larger than a hemp-seed to concretions of some dimensions. Their general outline is more or less round, or occasionally oval; but the surface is in the great majority of instances very rough and irregular, and hence they have been likened to a mulberry, though the irregularities are more often warty or thorny, or the oxalate may be deposited mainly in beautiful crystals, as in a case recorded by Dr. Church.[1] This superficial roughness causes much irritation, and leads to hæmorrhage. Hence oxalate of lime calculi are of a dark, greyish-brown colour, or even blackish-brown, owing to the admixture of altered blood-pigment. They are remarkably hard, and break into angular pieces when crushed. The fractured surface is almost always amorphous, and presents concentric laminæ. Chemically a mulberry calculus is characterized by being soluble in mineral acids, but insoluble in organic acids and alkaline carbonates. By the action of the blow-pipe it is first blackened, and ultimately reduced to lime, the oxalate being converted into carbonate, and the carbonic anhydride then driven off.

(*d*) *Phosphates.*—Primary concretions of calcic phosphates are not common. *Basic phosphate of lime* or *bone-earth* occasionally forms an entire calculus, or, in still rarer specimens, alternates with uric acid, as in one which is in the museum of the Manchester Infirmary, alluded to by Dr. William Roberts. Stones of this nature may be numerous. They differ much in size, but may attain that of an ordinary hen's egg. Their colour is white or yellowish-white; and the surface is smooth or roughish. They present different degrees of hardness, and their texture is either loose or compact. The broken surface always presents an amorphous, earthy, or chalky appearance. *Ammonio-magnesian phosphate* very rarely forms other than minute concretions, which are remarkably white, rough superficially, and of moderate hardness, the fractured surface being granular, yet crystalline and radiated. Renal calculi may consist of a mixture of *phosphates of lime* and *ammonio-magnesian phosphate*, constituting the so-called *fusible calculus*, though this variety is chiefly found in the bladder. As a primary concretion it is extremely exceptional, and is said to be chiefly observed in old people. The mixed phosphates may be deposited secondarily on a calculus retained in the renal pelvis, thus forming its superficial portion; and such a stone may grow to a very large size, and may become very irregular in shape. The surface may be studded with crystals of triple phosphate. Calculi of this nature are white and chalky-looking, of a loose and friable texture, and a section is either laminated or irregular. These concretions are insoluble in water and alkalies, but dissolve readily in mineral and other acids, though without effervescence, and they cannot be burned away with the aid of the blow-pipe, but under a strong heat fuse into a substance like enamel.

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[1] Pathological Transactions, vol. xx. p. 239.

(*e*) *Carbonate of Lime.*—Calculi composed of this ingredient are extremely rare in man, but are said to be not uncommon in herbivora. They were formerly supposed to be prostatic calculi, but their formation in the kidney has been undoubtedly established. Dr. William Roberts[1] quotes a case of his own, and also one of Dr. Haldane's, in which a large number of small concretions of carbonate of lime were found. Usually these calculi are very minute, being almost like gravel, and they are seldom larger than a hazel-nut. They are generally spherical, smooth on the surface, of variable colour—yellowish, grey, or bronze-colour, sometimes with a metallic lustre, and usually very hard. They are easily soluble in mineral acids, with effervescence, in consequence of the escape of carbonic anhydride; acetic acids acts slowly upon them, dissolving the carbonate, and leaving behind an animal matrix; they are not affected by caustic potash; and their surface becomes covered with a brilliant iron-grey enamel when they are heated to whiteness before the blow-pipe.

(*f*) *Cystine.*—This form of concretion is also very rare, but when it does occur it is usually made up entirely of cystine. The size and weight vary much; and the calculi are generally elongated, being cylindrical or egg-shaped. The specimens already alluded to, which were exhibited before the Pathological Society by Dr. Leared, ranged in size from that of a millet-seed to a flattened pea, but were rough and irregular in shape. One shown by Dr. Church,[2] removed from the pelvis of the right kidney of a man, aged fifty, was very irregularly-shaped, and weighed, when dry, 59 grains, after some had been used for analysis. Cystine calculi are of a dull-yellow colour, which frequently, but not invariably, changes to a pale emerald-green on exposure to the air; and the surface is lustrous or covered with crystals. They are easily friable, and can be scraped with the nail. A section presents a radiated structure, with a certain degree of transparency and brilliancy. Cystine calculi are soluble in fixed alkalies and their carbonates, in mineral acids, and in ammonia. From the latter solution characteristic six-sided crystals of cystine are deposited on evaporation after exposure; it may also be recognized by its chemical tests. Calculi made up of cystine yield a large proportion of sulphur.

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[1] Urinary and Renal Diseases, third edition, p. 283 *et seq.*

[2] Pathological Transactions, vol. xx. p. 240.

(*g*) *Xanthine.*—This constitutes one of the rarest varieties of calculus, specimens being mere curiosities. A stone composed of xanthine is light or dark brown, or deep yellow, smooth, and hard. It may be partly bright, and on rubbing assumes a waxy lustre. Its size and weight vary widely, the concretion in most recorded instances having been very small, but there is a specimen in the Museum of the College of Surgeons weighing ninety grains, and Langenbeck removed a xanthine calculus as large as a small egg. This substance was first discovered in a stone by Dr. Marcet in 1817. It is soluble in alkalies, but not in carbonate of potash. It also dissolves in dilute nitric acid without effervescence, assuming a golden yellow colour, and on evaporation a bright yellow residue is left.

(*h*) *Fibrin and Blood-clots.*—Concretions of this kind originate in renal hæmorrhage. They are usually small, and the blood being partially decolorized, they have a reddish-brown, yellowish-brown, or dirty white colour. They are sometimes soft, but generally firm, elastic, and tough, the consistence in some recorded specimens resembling that of bees-wax. It must be remembered that the remains of a blood-clot may form the nucleus of other calculi.

(*i*) *Mixed.*—Calculi made up of different constituents are not uncommon, their characters being accordingly modified more or less. Thus the nucleus may be different from the body, for instance, a nucleus of oxalate of lime may be present in a uric acid stone, or, more rarely, the contrary is observed; again, the body may consist of alternate layers, more or less complete, of oxalate of lime and uric acid or urate of ammonia, such an arrangement forming what is termed an *alternating* calculus; and, lastly, any stone may be covered with a crust of phosphates.

3. *Direct effects on the Renal apparatus and neighbouring structures.*—It is only practicable under this heading to indicate briefly the morbid conditions which renal calculi are liable to set up under different circumstances.

These concretions may be formed in both kidneys, and they may be of a different nature on the two sides. It has been affirmed that the left organ is more frequently affected than the right. The direct lesions induced by stone are usually only observed on one side, the opposite kidney being, however, frequently affected indirectly. It must be remarked at the outset that in many instances a calculus does not produce any notable effect upon the renal structures, either because it escapes along the ureter into the bladder soon after its formation, or because it becomes more or less encapsuled or encysted, and thus is rendered incapable of producing further mischief. The consequences of renal calculi depend upon their number, situation, size, shape, superficial characters as to smoothness or roughness, density, the time they remain in any part, and other circumstances. In general terms the lesions induced may be said to be the result either of obstruction to the escape of urine or of morbid products from the kidney; owing to the lodgment of a stone in the excretory passages; of mechanical injury; or of irritation setting up some form of inflammation. They may be most conveniently described according to the several parts of the renal apparatus which the concretions directly implicate.

(*a*) *Kidney.*—It may be mentioned that deposits are not uncommonly found in the renal tubules, consisting of similar substances to those which enter into the composition of certain calculi, and especially urates, and these are liable to set up chronic changes in the kidney, so that the organ becomes granular, contracted, and indurated, a condition typically illustrated by the "gouty kidney." If one or more calculi remain in the substance of the organ, they cause more or less irritation, and this may lead either to congestion and hæmorrhage, to the development of a surrounding fibrous cyst, or to the formation of one or more abscesses, which may burst into the renal pelvis and be discharged by the ureter, or may open in some other direction. In exceptional instances the renal tissue becomes actually gangrenous. The kidney becomes often seriously affected, as the result of the impaction of a stone in its pelvis or ureter.

(*b*) *Infundibulum and Pelvis.*—It is a common occurrence for one or more stones to lodge, either for a time or permanently, in this portion of the renal apparatus. They are liable to injure the mucous lining, causing more or less laceration, with consequent hæmorrhage, and the discharge of urine mixed with blood. This effect is particularly liable to happen when a calculus is disturbed by walking, running, riding on horseback, or other modes of exercise, and it may only occur from time to time. As in the substance of the kidney, so in its pelvis, a stone may become more or less surrounded by a fibrous pouch, and be thus rendered innocuous, as in a case already alluded to, recorded by Dr. Church in vol. xx. of Pathological Transactions.

In rare instances a calculus which remains in the renal pelvis goes on enlarging until it completely fills this space, and occasionally even the infundibulum and calices. Hence the urine which is formed is dammed up, and cannot escape. A portion of it is re-absorbed, but the remainder gradually accumulates behind the calculus, giving rise to the condition termed *hydronephrosis.* By degrees the renal tissue becomes atrophied in consequence of the pressure exerted upon it, its papillæ first becoming flattened, and ultimately nothing may be left but a fibrous sac containing altered urine, with perhaps a trace of kidney-structure on its walls, and showing partial partitions in its interior with spaces between, corresponding to the dilated calices. The obstruction may be subsequently removed, so that the fluid escapes, and the sac then shrivels up to a very small size. By far the most frequent result of calculus retained in connexion with the kidney is the development of *pyelitis.* This condition is indicated at first by injection of the membrane, which may become of a deep red colour, and numerous minute extravasations of blood are sometimes visible. The epithelium is shed more or less abundantly, and is discharged with the urine. Later on a muco-purulent or actually purulent fluid is formed, of a whitish or yellowish colour, and more or less turbid and thick. It may be seen bathing the surface of the lining membrane, and often covering the calculus, or mixed with urine, stones, or their *débris*, or calcareous matters may be precipitated; at this time the membrane becomes paler and is variously discoloured. Occasionally a fibrinous deposit is observed on its surface, or it may be ulcerated or even gangrenous. Should the escape of the inflammatory products be prevented by the impaction of a calculus, they accumulate and distend the pelvis and infundibulum constituting the condition termed *pyonephrosis*. If the obstruction is permanent, the kidney becomes involved and undergoes a purulent degeneration—*pyelo-nephritis*—ultimately becoming completely disorganized, merely a sac remaining, containing pus or calculi or their *débris*. This may attain considerable size, and frequently form extensive adhesions, while its walls consist of the thickened capsule of the kidney with some remnants of renal tissue, and its cavity may be more or less sacculated. Ultimately it may rupture in various directions, such as externally in the loins, into the colon or duodenum, into the peritoneum, or into the thoracic cavity. In some instances the obstruction to the escape of the fluid is only temporary, and it may occur from time to time, so that the sac fills up and is emptied at intervals.

(*c*) *Ureter.*—A calculus from the kidney frequently passes along the ureter to the bladder without doing any material damage. One serious event which may occur is that it becomes impacted, so as to obstruct the tube completely. This is followed by one or other of the consequences in connexion with the kidney already described, but in exceptional cases it seems to lead merely to atrophy of this organ. The ureter above the seat of impaction generally becomes dilated and thickened, while below it may become narrowed. A stone may also lead to inflammation, ulceration, or even gangrene of this duct, and the subsequent cicatrization of an ulcer may close up its channel permanently. Lastly the ureter may be much thinned at the seat of impaction, or actually ruptured.

(*d*) *Surrounding structures.*—The tissues immediately surrounding the kidney may become the seat of inflammation—*perinephritis*—terminating in the formation of pus. This may happen from the mere extension of irritation from the kidney, or as the result of the rupture of an accumulation of pus in the kidney or its pelvis into this part. Should an abscess form it may open in the various directions already indicated. Dr. Cayley[1] has recorded a case in which a calculus was discharged from an abscess in the right lumbar region, and subsequently two small calculi ulcerated out, one close to the crest of the ilium, one below the great trochanter.

4. *Remote effects and Complications.*—Associated with renal calculus there may be various morbid conditions in other parts of the body, depending upon the presence of a gouty diathesis. If one kidney is destroyed or otherwise affected, so that it cannot perform its functions,

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[1] Pathological Transactions, vol. xxvi. p. 128.

the opposite organ tends to become hypertrophied, so as to compensate for the loss of the secreting structure. An important result of renal stone, if it should cause the formation and discharge of much pus, is that it leads to the development of albuminoid disease, which affects the opposite kidney, as well as other abdominal organs or other structures. This condition may also cause septic poisoning, followed by pyæmic abscesses in various parts. The consequences of the rupture of a purulent collection in connexion with the kidney will depend upon the direction which the pus takes, and in this way peritonitis, pleurisy, and other serious lesions may be set up.

SYMPTOMATOLOGY.—The symptoms which may be anticipated in cases of renal calculus are referable to the following causes:—(1) Functional disorder of the kidneys; (2) mechanical irritation or injury set up by the stone; (3) the passage or attempted passage of a calculus along the ureter to the bladder; (4) the local pathological conditions which it may originate, especially calculous pyelitis and its consequences; (5) the general or constitutional disturbance, and the remote lesions which result from these pathological conditions.

The exact symptoms which may be thus originated, and the clinical progress of the disease, vary considerably in different cases, but in general terms the local phenomena may be summarized as pain or uneasiness in one or both renal regions; a tendency to frequent micturition in some cases; one or more attacks of renal colic; morbid conditions of the urine, including hæmaturia, the presence of epithelium-particles from the infundibulum and pelvis, of fragments of calculous matter, or of more or less pus; and the development of a renal tumour, due to the accumulation of fluid as the result of obstruction by a calculus, which may be permanent or only observed at intervals, frequently presents a sensation of elasticity or fluctuation, and is liable to open in various directions, the resulting symptoms differing accordingly. The general system also commonly suffers to a greater or less degree; and other organs besides the kidney may become involved as the result of complications.

Before considering the clinical history of calculous disease of the kidney in detail, attention must be drawn to certain important facts which relate to this subject. In the first place it is a matter of frequent observation that a renal concretion may remain in the kidney for an indefinite time without any indication or symptom of its presence,—indeed there are many instances in which a calculus has been found lodged in the kidney, packed as it were securely in one of its calices or in a pouch, without any known indication of its existence throughout a long life. In the Westminster Hospital Museum there is a specimen illustrating this. Dr. Dickinson in the Pathological Transactions, vol. xxi. p. 255, has described a case of calculous pyelitis in which no symptoms of any distinctive character, or with any special reference to the renal organs, occurred till two years before the patient's death, and then only a sediment in the urine, like pus, but there was no disturbance of the general health, no pain or inconvenience, no frequency of micturition, nor had there even been pain or uneasiness referred to the course of the ureter. The left kidney had been converted into a large cyst, containing nearly a pint of purulent fluid. The cavity contained many minute concretions of oxalate of lime, not larger than poppy-seeds.

In other instances the symptoms are of a very obscure nature, there being, perhaps, only a little uneasiness or sense of aching over one lumbar region, either continuous or occurring from time to time, especially after effort, or a tendency to frequent micturition. It may happen that even serious morbid changes may be set up in the kidney insidiously, without the occurrence of any prominent symptoms to draw attention to the presence of calculous disease. Or again, symptoms may be sufficiently characteristic for a time, but subsequently cease, owing to the stone becoming encapsuled, and thus being prevented from doing further mischief.

Again, observations have established the fact not only of the absence of all symptoms indicative of the existence of a calculus in the kidney, but even its escape and its descent down the ureter, its presence in the bladder, and its passage through the urethra, may all occur without pain or disturbance of any kind, except that during micturition a sense of some slight obstruction to the flow of urine is followed by the click of something solid falling into the chamber-vessel, and a small concretion of uric acid is seen, round and smooth. Such cases are not infrequent; and of some the writers have personal knowledge.

In other patients no symptoms of the formation of such a concretion or of its presence, no disturbance or alteration in the characters of the urine, to lead to the suspicion of such a disease, are manifested, till a sudden attack of renal colic occurs, or this may come on in cases in which a calculus is known to be present in the kidney, or the patient may even be subject to attacks at intervals. The colic is due to the entrance of the stone into the ureter, and if this passes along to the bladder, the symptoms become aggravated during its progress, but it must be remembered that the calculus may only attempt to escape, afterwards returning to the pelvis of the kidney. They are mainly due to the retention of urine behind the obstruction, with consequent dilatation of the passages; and to the spasmodic peristaltic action excited in the walls of the ureter, with the object of expelling the stone. The attack may set in quite spontaneously, even when the patient is asleep in bed; or it may depend upon some bodily exertion or jolt. It is characterized as follows:—Intense pain is felt in one lumbar region, often of a sharp, cutting, or pricking character, speedily becoming most excruciating, and accompanied with a severe griping colic-like paroxysm over the abdomen. The pain shoots in various directions,—downwards along the ureter to the hypogastrium, across the abdomen, along the crest of the ilium, directly backwards, or upwards towards the chest. Pain is also frequently felt at the end of the penis, along the inside of the thigh, and in the testicle on the same side, this organ being also retracted, and in course of time becoming in many cases tender and swollen. The leg may feel more or less numb. Accompanying these sensations there is usually a constant desire to micturate, along with strangury, only a very small quantity of urine being passed, drop by drop, which causes much burning pain along the urethra, and which is usually high-coloured, often containing more or less blood, mucus, or pus. The urine may, however, be quite natural in appearance and clear, if the ureter is completely blocked up, and the excretion comes from the healthy kidney. Gastric symptoms are frequently prominent, and may be very distressing, namely, nausea, retching, or vomiting, which may be bilious. The patient is of necessity in much distress, and while very restless and anxious, dreads to move, and lies doubled up with the thighs flexed, or presses upon the affected side. In most cases there is more or less general depression, and often the patient becomes utterly helpless and prostrate, while signs of collapse appear, with a feeling of faintness, coldness of the surface, clammy sweats, and a rapid, small, and very feeble pulse. In some cases actual syncope occurs; or there may be general tremors, spasms of various muscles, or general convulsions. The respirations are frequently hurried. In prolonged cases more or less pyrexia is set up. Even death may result from the intensity of the suffering and its consequences. In pregnant women an attack of renal colic usually leads to abortion.

The severity of the symptoms just described varies in different cases, and remissions followed by exacerbations are observed during the passage of the calculus. A sudden subsidence of the paroxysm, preceded by a marked exacerbation, usually indicates the entrance of the stone into the bladder, accompanied with a sense of intense relief, and the patient may be conscious of something falling into the bladder. The time taken to accomplish the transit, and the consequent duration of the attack, also differs much, ranging from a few hours to days or weeks. Occasionally the paroxysm subsides gradually, in consequence of the stone crumbling down, and being discharged in fragments. In exceptional instances it becomes permanently impacted, leading to the development of hydronephrosis; or it may lead to ulceration and rupture of the ureter, followed by fatal peritonitis. As a rare event, complete suppression of urine occurs, followed by uræmic symptoms and death. This may happen either from calculi being passed on both sides simultaneously; from one kidney being diseased, and the stone escaping from the healthy organ; or in exceptional cases even when the obstruction is on one side, while the opposite kidney is perfectly healthy, and its ureter entirely free. When an individual is subject to repeated attacks of renal colic, they often diminish in severity, owing to the ureter becoming dilated.

When a calculus reaches the bladder, the subsequent course of events is again liable to vary. In favourable and not infrequent cases, the concretion escapes through the urethra with the urine. If of very small size, it may be expelled without difficulty, and almost without notice. In other instances an act of micturition is interrupted by a sudden stoppage of the stream, followed by straining, with anxious efforts to relieve the symptoms of retention, until a violent gush of urine projects the calculus from the urethra, and the sound of a solid substance falling into the vessel announces to the patient the spontaneous relief to his disease. In most cases, however, the stone remains in the bladder, increasing in size, and setting up cystitis or other lesions, unless it is got rid of by surgical interference.

The differences in the character and intensity of the symptoms of renal colic, and in the subsequent progress of events, clearly arise from the size, shape, external characters, and composition of the calculus. Thus a small concretion is more easily voided than a large one; and one of uric acid than one of equal size composed of oxalate of lime. If the urine can trickle down by the side of the stone, the symptoms will be less severe than if the ureter is completely blocked.

In another class of cases the first indication of renal disturbance is the passage of a small quantity of blood or blood-stained urine, and this occurrence is chiefly associated with, and apparently excited by, some bodily effort. A jump on horseback in the hunting-field, a jar or concussion in a carriage over rough ground, or some such accidental occurrence, is followed by the appearance of blood in the urine; or the hæmaturia may occur without any such physical act.

The amount of blood passed is seldom considerable, although the urine may be blood-stained, and the microscope detect blood-corpuscles for several days afterwards. Sometimes the attack of hæmaturia is accompanied by lumbar uneasiness—a sense of aching in the loins rather than pain. There may be frequency of micturition, and pain at the neck of the bladder and extremity of the urethra, relieved by passing urine. The more typical symptoms of pain in and retraction of the testicle, numbness in the course of the external crural cutaneous nerve, or pain darting from the kidney through the ureter, may not be present at the early development of the renal symptoms.

Attacks of hæmaturia may occur every three or four weeks, the general health remaining unimpaired, but with obscure indications of renal irritation. In a case observed by one of the writers, hæmaturia occurring at intervals was the only symptom present for a long time.

The microscopic and chemical characters of the urine in these, at the outset, somewhat obscure cases, seem of the greatest diagnostic value. The urine will, during the presence of blood in it, be slightly albuminous, but the albumen disappears altogether when the hæmorrhage has ceased, so that in the intervals, supposing them to extend to several weeks, the urine will be perfectly free from all trace of this ingredient. The sediment, when examined by the microscope, will be seen to consist of isolated blood-discs, and a few ordinary mucus-corpuscles; later, however, after several of these attacks of hæmaturia, the proportion of the mucus or exudation-corpuscles will greatly increase, and it frequently happens that amorphous semi-crystalline masses will be seen, either free or buried in blood-clots, or occasionally surrounded by a group of exudation-cells.

Corresponding to these changes in the microscopic characters of the urine there will be observed subjective symptoms more especially typical of renal irritation, namely, more or less lumbar pain, with intervals of aggravation, extending down the ureter of the affected kidney to the neck of the bladder; uneasy aching sensations referred to the crest of the ilium, with numbness or altered sensibility in the cutaneous nerves of the thigh; a drawing-up of the testicles; and greatly increased irritation at the extremity of the urethra, leading in many cases, in both men and women, to a most distressing desire to pass water, but a scanty amount being discharged at each act. The urine now presents a great increase in the proportion of mucus, so that it falls to the bottom, instead of, as in healthy urine, half floating midway as a cloud.

The line of demarcation between calculous nephritis and calculous pyelitis is sufficiently marked in typical cases, but these are not so frequent as may be supposed; for in the majority of this class of renal disorders the first form passes by imperceptible degrees into the second, and for all practical purposes of treatment the two may be considered as the same. Pathologically, however, it is desirable to consider both forms as distinct, as the termination and ultimate results differ materially.

It may be stated that if to the symptoms of a paroxysm of nephralgia, there be added occasional attacks of hæmaturia, we have the typical indications of calculous nephritis. It must be remembered that in the intervals of these paroxysms, and after a lapse of a few days from the attack of hæmaturia, the urine returns to its ordinary healthy aspect, the only deviation from health being some divergence in the normal relative proportions of urea and uric acid.

A stone in the kidney, as has been already observed, may become permanently lodged in the seat of its origin, being surrounded by a capsule of areolar tissue, which screens the organ from further irritation, and also prevents any further addition to the growth of the calculus. This, however, rarely happens. Usually the stone is detached from its original seat, and a larger area of irritation is thus excited. Fresh material is added to its surface, and with this increased growth increased embarrassment in the urinary outlets occurs, giving rise to calculous pyelitis and its consequences. These changes are chiefly—it may be said solely—recognized by the altered qualities of the urine.

The progress of calculous pyelitis may be characterized by three groups or epochs of symptoms:—

1. Special alterations in the qualities and aspect of the urine.
2. The development of a renal tumour.
3. The formation and detection of a renal abscess.

It cannot be too forcibly observed, that the pathological changes which take place in calculous disease of the kidneys are various in degree and in the rapidity of their progress, as well as uncertain as to the final results; for it can never be definitely predicted in any given case what the duration or termination may be.

The clinical history of calculous pyelitis may conveniently be traced from the symptoms of calculous nephritis, which in the great majority of cases are the precursors of the more extended malady. With varying intervals in different patients, after experiencing attacks of nephralgia with occasional indications of hæmaturia, the frequency of micturition increases permanently—so that the irritability of the urinary passages, which in the early stage occurred in paroxysms as it were of a day or two at a time, now becomes constant, the patient having to rise many times during the night, and passing water every two or three hours during the day. The urine, if examined, is found to contain a much larger proportion of mucus than in health. This urine, when set at rest, instead of showing a delicate floating cloud, composed of a few epithelial cells, occupies perhaps one-third or even one-half of the glass—it is, however, still flocculent and not sedimentitious—and is composed entirely of what are called mucus- or exudation-corpuscles or modified epithelium. The amount of these corpuscles is not yet sufficient to impart a cloudy appearance to the urine, until it has been allowed to settle in an appropriate glass. In a short time, however, they increase largely, so that the urine when passed is obviously cloudy, and appears just as ordinary urine would if a teaspoonful of milk had been added to it. This marks the first stage of the urinary change—the passing of a mucous urine into one more or less purulent.

The cause of these changes in the characters of the urine must be sought in the increased area and intensity of irritation which an increasing concretion is setting up. It is the irritation of a foreign body located in the urinary outlet.

The purulent character of the urinary sediment is marked by a most distressing frequency of micturition—often being most urgent—every hour or even every half-hour, during both day and night.

Urine thus charged with purulent elements has a special proneness to undergo molecular change. At this stage urates are usually largely in excess, and become copiously deposited on the exterior of the calculus.

It is a clinical fact of some significance, that although the urine in the progress of this disease becomes more and more purulent, yet it uniformly maintains an acid reaction, and preserves its complete fluidity, separating when set at rest into two distinct portions—a well-defined sediment of pus-corpuscles; and a supernatant, clear, slightly acid, and slightly albuminous fluid urine. These conditions mark the limitation of the diseased action to the pelvis of the kidney. If the disease extends beyond the infundibulum and reaches the bladder, a very characteristic change in the quality of the urine takes place. The urine is passed ropy and ammoniacal, and deposits copiously crystals of triple phosphate. It is of much importance to decide how far, or if at all, the bladder has become implicated. This can only be etermined by an examination of the urine immediately after mic- urition. For it may happen, according to the season of the year, e temperature of the air, or the number of hours after excretion, at a urine derived from a kidney the seat of a simple calculous yelitis, will assume all the properties of a urine passing through a ladder the seat of disordered action. That which in a purulent urine, ossessing when voided all the characters above enumerated as dia- nostic of calculous pyelitis, will very shortly become by decomposition mmoniacal, ropy, and cloudy, and will exhibit numerous crystals of he triple phosphate.

If the urine be voided cloudy and turbid, with more or less viscidity, lkaline, and possibly ammoniacal, and if, instead of a well-marked pus sediment, there be a jelly-like magma; if the antecedent history of the case be that of calculous nephritis, then it may be confidently inferred that the original disease has become calculous pyelitis, with extension of irritation to the bladder itself. The cause of this extended disease for the most part arises from small portions of the recently deposited layers of the renal concretion becoming detached, and while present in the bladder setting up an irritation which induces the changes in the urine just enumerated. Proof of this is frequently met with when the jelly-like magma is examined, either with a magnifying glass or with the microscope; there is seen entangled in the mass amorphous matter, chiefly urates, embedded in it. The gelatinous material in which the concreting matter is concealed is a mucus ormed in the bladder in great excess, owing to the irritation of the material derived from the renal calculus. In these cases so abundant ftentimes is this gelatinous material, that the chamber-pot becomes ncrusted with a thick coating, which can be scraped off in masses, nd when chemically examined it is found to consist chiefly of urates nd uric acid grit.

It is at this stage that the possibility of these masses, or of granules uric acid, becoming the nucleus of a vesical calculus, presents itself. any of the symptoms at this stage point to this disease, and the xamination of the bladder by a skilful surgeon becomes desirable.

The microscopic objects in these urines are exudation and mucus orpuscles, isolated or in groups. Large fields of them are often seen, urrounding fine granular matter, or amorphous concretions of uric cid. Oval and pyriform cells from the pelvis of the kidney and reters may in the earlier stage be also visible.

It is in such cases as these, when the disease having originated in the idney has extended and eventually implicated the bladder, that the fferential diagnosis between renal calculus and stone in the bladder ecomes difficult, though important, and indeed can only be determined y a careful examination of the bladder in the usual way for stone.

Another class of cases will terminate in the formation of a renal umour and renal abscess, the passage of the pus generated within the avity of the kidney becoming obstructed by the growth of the calculus, hich becomes branched and moulded into the hollows of the calices, or impacted in the infundibulum, while it increases by the accretion of urates and triple phosphates, and eventually so blocks the outlet into the ureter as to retain both urine and pus within the diseased organ. The recognition of this stage of the disease is not difficult. The chief and most prominent feature is the varying aspect and condition of the urine. The impaction of a calculus in the cavity of the infundibulum causes, as has been already shown, an accumulation of the urine secreted by the kidney, and of the pus generated by the diseased structures behind the obstacle; a dilating force is thus created, which leads to the dilatation, sacculation, and eventually to the complete atrophy and obliteration of all secreting tissue. At first, however, the retention of the fluid is not complete. The calculus does not completely fill or obstruct the outlet; the dilating force of the accumulating fluids suffices to expand the infundibulum sufficiently to permit a portion of them to pass down the ureter into the bladder. This stage of the disease is markeo therefore by the fluctuating character of the urine, and by certain periodical exacerbations of the patient's sufferings.

The order of these occurrences is very much as follows:—A certain sense of uneasiness, fulness, or aching is perceived in the lumbar region on the side affected. The urine is noticed to become daily less milky or impregnated with pus; it gradually acquires an ordinary appearance, and the patient would flatter himself that his complaint was slowly disappearing, but that the pain and aching in the back augments almost in proportion to the apparent improvement in the urine. A careful examination of the region of the kidney demonstrates the presence of a renal tumour, which affords more or less evidence of elasticity or fluctuation.

In these cases it must be supposed that one kidney is free from disease, so that while the secretion of the one is retained, the function of the other continues uninterrupted. It is not usual, and only happens in rare cases, that both kidneys are simultaneously affected with calculous disease. Museums, however, contain specimens of such, and the experience of physicians is cognizant of such cases. It is, however, only as the calculous disease advances, and has existed for many years, that the second kidney becomes implicated. One of the writers (Dr. Basham) has had the opportunity of studying and keeping under observation for twenty-two years this form of double renal calculus. The disease in the left kidney was determined at the age of fifty-seven by the formation of a renal tumour, and the subsequent excretion for some eight or nine years of a purulent urine. At the age of sixty-five, eight years after this calculous disease of the left kidney was diagnosed, a tumour in the right lumbar space, fluctuating, and by pressure imparting pain in the course of the ureter of that side, with subsequent diminution of the area of the tumour, and greatly increased amount of pus in the urine, pointed to the implication of the right kidney in a similar form of disease. The patient lived to the age of seventy-six, and a post-mortem examination demonstrated the all but complete destruction of both kidneys, which were crowded with sacculated pouches, the cortical part of either kidney not exceeding in thickness the rind of an orange, and both infundibula being filled with accumulations of concretive matter composed of triple phosphates and urates, with central layers of uric acid and oxalate of lime. The ureters for some half-inch down were packed with this calculous *débris*.

As the renal tumour increases there is often a great amount of gastric disturbance, in the form of nausea, retching at times, and great inappetency, with absence of sleep; and these conditions frequently alternate remarkably with the retention of the fluids within the renal abscess. As this increases these constitutional symptoms increase also; but as soon as the outlet for the escape of pus and urine has been again established, the disturbance ceases, and the patient returns to a state of comparative ease and health. It may, therefore, in these cases be confidently expected that so long as the urine is purulent (but not ropy), there is a comparative exemption from suffering; but when it clears and presents little to notice, the patient's sufferings return.

Sex appears to exercise a modifying influence, as women endure this disease much longer, suffer less distress, and escape many of the severe complications which present themselves in men. The bladder less frequently participates in the diseased state of the urethra; this probably arises from the freer passage through the urethra of calculous particles or concretions in women than in men, and the former sex also escape the distress, trouble, and suffering occasioned by the presence of prostatic disturbance.

The general system suffers more or less during the progress of the symptoms above described, as indicated by pyrexia, wasting, weakness, loss of appetite, and other phenomena. The course of the symptoms towards a fatal termination in cases of calculous pyelitis varies much in different constitutions. In some the appetite, as well as the digestive and assimilative functions, slowly fail, a distaste for food of any kind exists, and a low adynamic fever is the result, with a quick and feeble pulse, a brown dry tongue, the skin being dry, but not much elevated in temperature, the stomach often becoming irritable and rejecting all ingesta, and thus the patient slowly sinks, and eventually dies from asthenia. Some cases are complicated with symptoms of uræmic poisoning; and in others, particularly those whose bladder complication has for some time pre-existed, with ropy muco-purulent turbid urine, the termination of the case is marked by symptoms of some form of capillary embolism, developed by a purulent-infected blood, the locality of this complication being uncertain and variable. These low forms of what at first appears like erysipelatous inflammation may involve one leg or foot, with extension of the swelling and hardness up to the groin—symptoms beginning with indications only of capillary obstruction, but terminating with evidence of fibrinous exudation in the sheaths of the large vessels of the thigh. In other instances the evidence of the pus-poisoned blood rests on the development of pulmonary symptoms, dyspnœa, and exhaustion. In all, however, whatever form of complication may be developed, the same defective assimilative power, inappetency and rejection of food, with a weak, small, but frequent pulse, a dry tongue, and gradual exhaustion, invariably mark the termination of the case.

Various symptoms may arise during the progress of calculous disease of the kidney, from the rupture in different directions of a hydronephrosis, of a purulent collection in the kidney, or of a perinephritic abscess. Thus a collection of pus may make its way to the surface, either in the lumbar region or elsewhere, finally opening or being artificially opened, and then perhaps discharging calculi or their remains, as in the case previously mentioned which is recorded by Dr. Cayley. After such a termination recovery usually follows. Or the rupture may take place into the peritoneum, with the usual symptoms of such an event, followed by those of peritonitis. Again, the fluid may find its way into either the ascending or descending colon, according to the side affected, this event being indicated by a sudden severe attack of diarrhœa, the discharged matters being mixed with pus, and the simultaneous subsidence of the enlargement. On the right side it has in rare instances communicated with the gall-bladder and duodenum. This communication with the intestine may terminate in recovery, but it is not nearly so favourable as the external opening. In exceptional cases the accumulation of pus penetrates through the diaphragm into the pleura, setting up the clinical signs of pleurisy; or it may open into the lung, and thus into the bronchi, being afterwards expectorated.

Lastly, it must be remembered that complications may be associated with renal calculus, either as more or less remote consequences, or as co-existing conditions; and they give rise to their several clinical phenomena. Thus, there may be symptoms of gout; or evidences of albuminoid disease of the healthy kidney and other organs may be developed; or unquestionably pulmonary phthisis may be set up as a result of chronic suppuration. Biliary calculi are sometimes associated with renal calculi, and one of the writers had a case recently under his care, in which the patient passed first a large gall-stone, and afterwards a renal calculus, consisting probably of uric acid.

DIAGNOSIS.—There are several points relating to the diagnosis of calculous disease of the kidney which it will be necessary to consider.

The first matter to be determined is the presence of one or more calculi in some part of the renal apparatus. This may be clearly indicated by the local sensations, combined with hæmaturia, or with other morbid characters afforded by the urine, evidencing the existence of calculous pyelitis. In doubtful cases, which are not uncommon, where the symptoms are obscure, very careful examination of the urine is demanded, for the detection of traces of blood, or of epithelium detached from the pelvis and infundibulum of the kidney; and it will be well to carry this out after the patient has made some effort. It must be remembered, also, that renal calculus may be clinically latent, and then it will be impossible to recognize this disease. Repeated hæmaturia, provided that the blood can be shown to come from the kidney and endemic causes can be excluded, will probably be due to renal calculus, even though there be no other symptoms of the disease.

Supposing that the diagnosis of calculous disease of the kidney has been established, it will in the next place be desirable to determine the nature of the stone, as this may have a material influence as regards the treatment. In many cases there are no adequate data for forming any definite and satisfactory conclusion on this matter, and at the best the diagnosis must usually be doubtful. It is founded upon the known relative frequency of different kinds of calculi; the constitutional condition, as affording evidence, for instance, of a tendency to the excessive formation of uric acid or urates; and upon the composition of the urine, or the presence in this fluid of gravel, of fragments of calculous matter, or of different crystalline deposits, as these elements will probably correspond to the constituent which forms the calculus.

The next point, which is of considerable importance, is the diagnosis of renal colic, due to the passage of a stone along the ureter. In a considerable proportion of cases this is perfectly easy, the symptoms already described being highly characteristic. Of course when there are no symptoms, in consequence of the small size of the calculus, its escape cannot be recognized. It must also be borne in mind that other objects may traverse the ureter besides a calculus, such as a blood-clot or a hydatid vesicle, and the symptoms characteristic of renal colic would then be observed to a greater or less degree. They may also be simulated by neighbouring affections, especially spasm of the lumbar muscles, severe neuralgia of the intercostal or lumbar nerves, spasm of the colon, and the passage of a gall-stone. In these conditions the urine is not altered. In the complaint last mentioned, the pain is on the right side, and is higher up in the hypochondriac region as well as more towards the front than in renal colic, while it is usually associated with, or followed by jaundice and other symptoms which accompany hepatic colic. Among the curiosities of medical literature, cases are described in which a communication has been established between the gall-bladder and the ureter, and hepatic calculi have consequently escaped into the urinary bladder. This course of events would generally be recognized by the presence of a large quantity of bile in the urine, apart from jaundice; and by the composition of the concretions.

Should the stone escape either into the bladder or through the urethra, it will necessarily be a matter of much moment to determine whether this is the only concretion, or whether one or more remain behind in the kidney. This question is often very difficult to decide, but much assistance may be derived from the composition of the calculus, and from observing if the local symptoms subside after it has descended, and if the urine returns to its normal characters. Should this course of events not happen, the probability will be that irritation is still kept up by remaining concretions, either in the same or the opposite kidney, of which the number cannot be determined. This is not always the case, however, for a calculus may produce lesions which are permanent, although the primary source of irritation is removed. It has been affirmed that when a number of concretions collect in the renal pelvis, it may be possible by manipulation to bring out a sound due to their being rubbed against each other.

It is always a matter of great consequence to make out whether calculous disease is confined to one kidney, or implicates both organs. The seat of the local sensations is of course mainly to be relied on for settling this point, these sensations being referred to the side affected. Assistance may also be derived from the nature of any concretion which has been passed. Thus a phosphatic stone is almost always unilateral, while uric acid calculi are not uncommonly found in both kidneys. In certain cases important information is derived from the characters of the urine. Should this fluid have been purulent, owing to calculous pyelitis, and should it become healthy and perfectly free from pus during an attack of renal colic, or in association with the formation of a tumour in one renal region, due to the blocking-up of the corresponding ureter, the great probability is that the opposite kidney is free from disease.

The diagnosis of the more prominent pathological effects produced by a stone in and around the kidney from other conditions is usually not difficult. Pyelitis is recognized by the characters of the urine; and any accumulation of fluid is followed by the development of an enlargement in the renal region, which presents a sensation of more or less elasticity or fluctuation. The alternation of purulent urine with a renal tumour presenting this character, as described under the symptomatology, is highly characteristic of calculous disease. The diagnosis of the exact cause of any local enlargement associated with this complaint is often a matter of much difficulty, but for practical purposes it may not be of much moment. The aspirateur may prove of much value in determining the nature of any fluid accumulation. The opening of such an accumulation in various directions must be judged of by the special symptoms which would then arise.

The last point which has to be noticed is the diagnosis of calculous disease of the kidney from other affections. In the first place this complaint has to be distinguished from neighbouring affections, especially lumbago and other painful maladies. Any persistent pain in either loin should be taken due notice of, and not merely referred, without proper investigation, to the muscles and fasciæ. The conditions likely to be mistaken for renal colic have already been pointed out. Then, again, calculous disease may be confounded with other renal affections, or with those of the ureter. It may be very difficult to distinguish it from mere gravel, which is liable to give rise to similar symptoms, though of a milder type usually. Cancer, hydatid disease, and other morbid conditions of the kidney may also be mistaken for calculus, especially as they may originate hæmorrhage, and the passage of blood-clots or hydatid-vesicles may simulate that of a stone. It must be borne in mind that calculous disease may exist along with these conditions. Pyelitis from tubercular disease or other causes may resemble that due to stone. Lastly, the relation of the kidney to the bladder is highly important. When a calculus is localized in the kidney, the symptoms occasionally seem to be referred to the bladder, even though this organ is quite healthy. Moreover, a stone or portions of it may pass into the bladder, and subsequently set up cystitis. This is usually indicated by the supervention of vesical symptoms, and particularly by the changes in the characters of the urine, which becomes ammoniacal, alkaline in reaction, and ropy. In some instances renal disease follows the vesical affection, but under such circumstances the characteristic symptoms of the latter have usually been sufficiently obvious in the early stages.

Prognosis.—Calculous disease of the kidney must always be regarded as a serious affection, and great caution should be exercised in forming an opinion as to how it may terminate in any particular case, or what may be the course of events. The progress is usually essentially chronic, and when death occurs, in the great majority of cases it takes place more or less gradually, as the result of direct or remote lesions induced by the calculus. In exceptional cases the fatal termination is rapid, in consequence of the bursting of a purulent accumulation, rupture of the ureter during the passage of a stone, uræmia from the blocking-up of this tube, pyæmia from purulent absorption, or other causes. The prognosis as to the cure of the complaint is worse in proportion to the time a calculus has remained in the kidney; to the extent of the lesions which have been set up; and to the presence of a constitutional condition, whether hereditary or acquired, favourable to calculous disease. It is also more unfavourable if the calculi are multiple, or if both kidneys are affected. On the other hand, recovery may be hoped for when the disease is recent, and comes under treatment at an early period; when it is of local origin; when the stone passes into the bladder, and there is no evidence that any more concretions remain in the kidney; and when the disease seems to be limited to one side. Even after extensive lesions have been set up, provided they are confined to one kidney, recovery may ensue, the involved organ becoming shrivelled up and useless, while the opposite one enlarges and assumes a compensatory activity. This may occur after the opening of purulent accumulations externally, or even into the intestines. It must not be forgotten that renal calculus may be got rid of for the time, while a tendency to the recurrence of the complaint remains, and a fresh concretion may form if this tendency is not removed.

TREATMENT.—The management of cases of renal calculus must be regulated in different cases according to the actual conditions present. There are, however, certain general principles or indications upon which the treatment has to be conducted, and these it will now be requisite to discuss.

1. *Preventive Treatment.*—It may happen that the chief aim of treatment should be to prevent the development of calculous disease under circumstances in which this complaint is liable to arise. This applies mainly to calculi of uric acid or urates, but the principle may also be recognized in connexion with some other varieties. The chief circumstances under which this preventive treatment is indicated are when there is a marked hereditary tendency to the disease, or the gouty condition is prominent, whether hereditary or acquired; when there is a history of calculi having been passed or removed on former occasions; when the urine presents certain abnormal characters, namely, that this fluid is persistently alkaline or ammoniacal, contains gravel of uric acid or oxalate of lime when first passed, or deposits crystals of either of these elements soon after its discharge and before it has had time to cool, or reveals the presence of cystine; and when there are signs of the existence of any renal disease which is liable to be followed by the formation of calculi. The preventive treatment must also keep in view the limitation of the further growth of a stone, or of the development of additional concretions.

In order to carry out this indication, general management is of the greatest importance in most cases, having reference especially to the diet and habits of life. The measures to be adopted are mainly those which have already been considered in describing the treatment of lithiasis and lithæmia. The diet must be duly regulated, being of a simple kind, while the amount of nitrogenized food, and especially of meat, must be particularly moderated. In some cases, indeed, it is desirable to forbid meat altogether, either permanently or from time to time; but this is by no means required as a rule, and a fair quantity may be allowed to those who take adequate exercise, and who lead an active life. Rich and indigestible kinds of food must be avoided, as well as all highly-seasoned dishes. It is very important that there should not be too long intervals between the taking of food, and also that large and heavy meals should not be indulged in: therefore the patient ought to have four or five light meals a day. When there appears to be any tendency to oxalate of lime calculus, vegetable substances which contain abundance of oxalates must be avoided, especially rhubarb and sorrel.

The question of drink has also an important bearing in the prevention of calculus. In the first place good water should be taken freely, and it is especially recommended, for certain physiological reasons, that a tumblerful be taken the last thing at night, and also about two hours before dinner. By this means a free and abundant flow of urine is maintained, the urinary ingredients are kept in solution, and any deposits formed in the kidney are likely to be washed away. Waters containing much lime had best be avoided. The effervescent waters—soda-water, potass-water, lithia-water, or seltzer-water are also useful in moderation. With regard to stimulants, great care must be exercised in their employment. In many cases they had better be avoided altogether, and all strong or heavy wines must be rigidly forbidden. In some instances a little well-diluted whisky or gin may be of use, by acting as a diuretic, or good claret may be taken with advantage.

In addition to these measures, it is usually necessary for any one who is prone to calculous disease to take more or less active exercise, and avoid a sedentary life; to maintain a healthy action of the skin by means of baths, followed by friction; and to wear warm clothing, with flannel next the skin. It is also desirable that the hours of rest in bed should be curtailed, as the recumbent posture seems to be favourable to the development of calculous disease, and therefore early rising should be practised.

Medicinal treatment is often of much service for the prevention of renal calculus. In the first place the functions of the digestive organs must be maintained in good order, the bowels kept well opened, and the secretions free. For these purposes alkalies or dilute mineral acids with some bitter infusion or tincture may be of service, with an occasional dose of blue-pill and rhubarb pill, as well as aperient mineral waters, especially the Hunyadi Janos or the Friederichshall water, taken the first thing in the morning, as frequently as may be required. Diuretics have been employed for the purpose of creating an abundant flow of watery urine, and thus dissolving or washing away any sediments which may have formed in any part of the kidneys. All powerful remedies of this class, however, ought certainly to be avoided, for they tend to irritate the urinary organs. The most important medicinal agents employed for the prevention of calculous disease are those which dissolve certain materials which tend to form concretions, and thus to prevent their deposition; or even dissolve them after they have been precipitated in the form of gravel. For deposits of uric acid, urates, and oxalates, certain alkaline salts are highly efficacious. Different salts of this class are recommended by different authorities, especially bicarbonate or phosphate of soda, bicarbonate or citrate of potash, and carbonate of lithia. It seems not improbable that these might severally be suitable for different cases, but in the writer's experience citrate of potash, in drachm doses twice daily, well-diluted, has proved generally serviceable. Certain mineral waters which contain alkaline salts are also of great value, such as those of Carlsbad, Vichy, Saltzbrunn, &c., some of which are useful at the same time on account of their aperient action. They must, however, be employed under proper supervision.

For the prevention of phosphatic deposits and those of carbonate of lime, certain acids are indicated, namely, carbonic acid water, and solutions of vegetable acids, such as citric, malic, or acetic, which in their passage through the system become converted into carbonic acid, and thus the urine is maintained in an acid condition. If the formation of secondary phosphates should result from retention and decomposition of urine in the renal pelvis, all that can be done is to try to promote a free flow of the excretion in an acid state, which might both wash out the space and neutralize the ammoniacal condition.

2. *Treatment to aid the solution or escape of a Renal Calculus.*—Bearing in mind the pathological consequences which may arise from calculous disease of the kidney, it is reasonable to conclude that as soon as there is evidence of its existence, the first object aimed at in treatment should be to get rid as soon as possible of any concretions and thus prevent further mischief. For this purpose attempts have long been made to dissolve renal calculi through the agency of medicines, which, by affecting the composition of the urine, might act chemically upon these concretions. This mode of treatment was, however, founded on a very unsatisfactory basis, until Dr. William Roberts,[1] by experiment and clinical observation, endeavoured to establish it upon a scientific and practical foundation. So far as renal calculi are concerned, it is probably only concretions of uric acid or urates which can be affected in this way, and these may be acted upon by alkalies under certain conditions. Dr. Roberts advocates this method of treatment when the urine is acid, and when the calculus is either known to consist of uric acid, or even when its nature is doubtful, as it does no harm should the stone be made up of oxalate of lime. He points out that the action of the alkalized urine must be continuous and incessant, and that a certain degree of alkalinity must be kept up. He prefers salts of potash, especially the acetate, in doses, for an adult of forty to sixty grains, for children twenty to thirty grains dissolved in three or four ounces of water; or the citrate, prepared according to the following prescription:—℞ Potassæ; bicarb. ʒxij. Acid citric. ʒviii. gr. 24. Aqum. ad. ℥xij. Of this solution the dose for an adult is six or eight fluid drachms, for a child three to six fluid drachms, mixed with three or four ounces of water. The dose should be repeated at intervals of not less than three hours, and it should be given with rigorous regularity during the waking hours. A dose should be taken the last thing before retiring to rest, and another in the course of the night, should the patient waken. Dr. Roberts urges the importance of frequently examining the *freshly-voided* urine when this plan of treatment is being carried out, and affirms that when this excretion is sweet, and has not undergone ammoniacal decomposition when first passed, there is no fear of the deposition of mixed phosphates upon the calculus, which has been one of the arguments against this mode of treatment. Should the urine become ammoniacal, the treatment must be suspended. Otherwise it may be carried on for months without doing any harm, and if the acetate or citrate is employed, the digestive functions are not at all impaired. The writer has recently had under observation a case in which there

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[1] See Medico-Chirurgical Transactions, 1865; and Urinary and Renal Diseases, Third edit. p. 299 et seq.

was strong evidence of the existence of a renal calculus composed of urates, and which seemed to be much benefited by this plan of treatment. Even if the calculus is not entirely dissolved, it may be diminished in size or broken up, and thus be enabled to escape by the ureter. The solvent treatment has also been attempted in cases of supposed fibrinous concretions.

Apart from the solvent treatment, there does not seem to be any plan open for aiding the escape of a calculus so long as it remains in the renal pelvis. If it should enter, or show a disposition to enter, the ureter, it appears advisable to encourage its passage downwards along this duct, even with its attendant suffering and possible consequences, rather than to try to cause the stone to return to its former seat. The late Sir James Simpson on two occasions practised successfully the plan of inverting the patient, so that the head was directed downwards, and then manipulating with the view of making the calculus fall back into the renal pelvis. Such treatment, however, seems only called for when the symptoms are very urgent and the concretion shows no disposition to descend. The descent of a calculus which is lodged in the ureter may sometimes be aided by careful manipulation and pressure in a downward direction, in the course of this tube.

When a calculus cannot be treated by solvents, and is too large to escape by the ureter, it has been suggested to keep the patient perfectly at rest in the recumbent posture for a prolonged period, in the hope that the concretion might form a pouch around it, and thus be rendered innocuous. In some cases certainly benefit might be anticipated from this plan.

3. *Treatment of Renal Colic.*—The symptoms which arise during the escape of a stone from the kidney to the bladder demand measures for their relief, and some of these measures will also assist in accelerating its descent. The patient should be kept as quiet as possible, and great benefit is often obtained from the employment of prolonged warm baths. The only reliable medicinal agents are narcotics and anodynes. Of these opium and morphia are the most important. The former may be given by the mouth in the form of the tincture or the liquor opii sedativus, in full and repeated doses, until the patient is brought well under its influence. In some cases, in consequence of sickness, it cannot be taken by the mouth, and may then be administered in starch enemata. Subcutaneous injection of morphia is most valuable in many cases. Belladonna and chloral are also useful remedies, and the latter has been successfully employed in combination with morphia. If the attack of renal colic is prolonged, the careful inhalation of chloroform from time to time may be resorted to. The use of watery diuretics has been advocated, with the view of creating a free flow of urine, and thus helping to drive on the calculus by the pressure of the fluid, but such a plan of treatment seems to be attended with considerable danger. Local applications may be of service, such as the assiduous

employment of hot fomentations, or the application of large warm poultices; to these anodynes might be added. Local removal of blood, by means of cupping, may be demanded in some instances, and in highly sthenic cases even general venesection might possibly be indicated.

4. *Treatment of Symptoms and Complications.*—The immediate pathological consequences of renal calculus not uncommonly demand attention, namely, hæmorrhage, with consequent hæmaturia, which must be treated by rest and the administration of astringents. Hydronephrosis, pyelitis, and purulent accumulations, either within or around the kidney must be managed on the same principles as when they arise from other causes, which are discussed under the several articles. Their remote effects may also require different modifications of treatment, such as that directed to peritonitis, pleurisy, albuminoid disease, &c. General treatment is also indicated frequently in the way of a supporting diet, stimulants, tonics, cod-liver oil, or other therapeutic agents.

5. *Operative Treatment.*—This may be required for the removal of an accumulation of fluid associated with calculous disease. Two special operations have been resorted to in some instances, namely, nephrotomy, and extirpation of the kidney. The former consists in making an incision into the kidney from the loin, and then extracting any calculi which can be discovered on examination. This operation is only admissible when an accumulation of pus has pointed externally, and it has become necessary to open this. Extirpation of the kidney has been practised for various conditions, and in a few instances with success. This operation has been advocated in certain cases of calculous disease, but few would venture upon such an heroic measure.

HYDRONEPHROSIS.

By Frederick T. Roberts, M.D., B.Sc., F.R.C.P.

Definition.—Hydronephrosis, (υδωρ, water, and νεφρον, a kidney), is the condition characterized by accumulation of urine within the pelvis of the kidney, due to some impediment interfering more or less with its escape. This leads to dilatation of the renal pelvis, infundibulum, and calices, with subsequent atrophy of the kidney-structure. The obstruction may be situated in any part of the urinary passages, from the upper opening of the ureter to the orifice of the urethra, and consequently the distension may be confined to the kidney, or may also involve more or less of the ureter, the bladder, or even the canal of the urethra.

Synonyms.—Hydrops renalis; Dropsy of the kidney; Hydrorenal distension; Hernia renalis; Hydronephron.

Etiology.—In a few exceptional cases of hydronephrosis which have been recorded, no cause of obstruction to the escape of the urine could be detected, but this was probably due either to its having existed at a previous period, and been removed; or to its having been overlooked at the post-mortem examination. In the great majority of instances the impediment can be satisfactorily made out, and the numerous conditions which have been found to originate hydronephrosis are conveniently arranged under the two groups:—1. Congenital. 2. Acquired.

1. *Congenital.*—A considerable proportion of cases of hydronephrosis belongs to this class. Out of fifty-two cases collected by Dr. William Roberts, twenty were congenital, thirteen of these being double.

When this condition exists at birth, it is often associated with various congenital malformations. Congenital hydronephrosis usually depends upon some abnormal state of the ureter. This tube may be greatly narrowed or even completely occluded in some part of its course, or at its entrance into the bladder. In other cases it has been found to communicate with the renal pelvis at a very acute angle, forming a valve-like arrangement, which prevents the escape of the urine, the difficulty being increased as this fluid accumulates in the pelvis. In very rare cases, as has been mentioned in a previous article, anatomical peculiarities affecting the renal artery may originate hydronephrosis, this vessel taking an abnormal course, or there being an additional artery, and in either case the ureter may be pressed upon, or the vessel may twist round it. Another congenital cause of hydronephrosis is closure of the canal of the urethra, which leads to dilatation of the bladder, as well as of both ureters and pelves of the kidneys.

2. *Acquired.*—Several important causes of hydronephrosis come under this category, and they may be thus arranged:—(*a*) Obstruction of the ureter or renal pelvis by the lodgment of a calculus or other mechanical obstacle, which blocks up the passage. (*b*) Disease of the walls of the ureter, leading to the contraction and ultimate closure of its channel. This may result from inflammation, ulceration with subsequent cicatrization, or carcinoma involving the walls of the ureter. (*c*) Disease of the bladder in the vicinity of the orifice of the ureter, closing up its outlet. An abscess of the walls of the bladder, and malignant disease, have been known to produce this effect. (*d*) Conditions affecting the ureter from without, either compressing it, dragging it out of its course and fixing it there, or bending it, and thus interfering with its channel. This class of causes is much more frequent in females than in males, and includes chiefly inflammation of the pelvic cellular tissue; peritoneal inflammation, originating thickened bands; retroflexion or prolapse of the womb; a pregnant uterus; cancer of this organ; and ovarian or other pelvic tumours.

ANATOMICAL CHARACTERS.—Hydronephrosis consists essentially in the gradual accumulation of the urinary excretion within the renal pelvis, this fluid in time becoming more or less altered in its characters; and of certain pathological consequences resulting from this accumulation In the majority of cases the condition is unilateral, especially if it becomes extreme, but it is not uncommonly double, though both kidneys are not usually affected to an equal degree. Out of fifty-two cases collected by Dr. William Roberts,[1] thirty-two were single (nineteen right and thirteen left), and twenty double. The degree of distension varies greatly in different cases. Thus the sac has been found to range from a size not larger, or even less than the normal kidney, to an enormous dilated space containing as much as thirty gallons of fluid. At first this fluid collects in the renal pelvis, infundibulum, and calices, but as it increases in quantity, it not only causes distension, but also leads to gradual wasting and absorption of the renal structures. The medullary portion first becomes affected, the papillæ being flattened, dwindling down, and finally disappearing altogether. Afterwards the cortical substance becomes involved, and undergoes similar atrophy, until ultimately but little or no traces of it remain. Even in very large hydronephrotic sacs, however, it is common to find portions of renal tissue on the walls, in the form of detached reddish islets, in which urinary tubules and Malpighian bodies can be detected on microscopic examination. In extreme cases no trace of kidney-structure remains. The sac usually presents a more or less lobulated appearance externally, and on opening it fibrous septa are found, corresponding to the external depressions, and partially dividing it into sacculi or chambers, which correspond to the dilated calices, and which communicate freely with the renal pelvis, and thus with each other. In extreme cases these septa disappear, and the sac becomes quite uniform and undivided. When the renal tissue has been removed, the walls of a hydronephrotic sac are formed of the thickened capsule, and consist of tough and dense fibrous tissue, highly vascular, smooth internally, but more or less rough and uneven externally, owing to adhesions having formed with surrounding structures. Occasionally plates of a fibro-cartilaginous tissue are found imbedded in the walls.

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[1] On Urinary and Renal Diseases. Third edit. p. 486.

The fluid which is found in cases of hydronephrosis almost always consists of urine more or less altered in its characters. It is very watery, of low specific gravity, often faintly alkaline in reaction, yellow in colour, and either clear or somewhat turbid. It usually contains the ordinary urinary constituents in various proportions, but urea is often only present in small quantity, and may be entirely absent. Uric acid is also occasionally wanting. There may be crystals of oxalate of lime. Admixtures of blood, pus, or epithelium may be found, and thus the fluid may present a reddish, reddish-brown, or greyish-yellow colour, as well as unusual turbidity. It is always more or less albuminous. In exceptional instances the contents of a hydronephrotic sac consist of a gelatinous colloid substance, as in a case reported by Dr. Dickinson;[1] of thick, fatty fluid; or of a caseous material.

Peculiarities are observed in exceptional cases, the hydronephrosis being limited to a part of the kidney, or there being two ureters, one of which is obstructed. If the seat of obstruction is in the course of the ureter or at its opening into the bladder, the duct partakes in the enlargement, and is found to be more or less dilated, elongated, tortuous, and thickened in its walls, the muscular element of which may be hypertrophied. The tube has been observed as large as the small intestine. When the urethra is closed, the bladder is also involved.

The effects of hydronephrosis upon neighbouring structures are to displace and compress them more or less, and these effects may be carried to an extreme degree, the abdominal walls becoming also stretched. The colon is usually situated in front of the sac, but occasionally is found on its inner or outer side.

SYMPTOMATOLOGY.—When hydronephrosis is congenital and double, should the child be born alive, death usually occurs within a very short period, varying from a few hours to three or four months; in exceptional cases, however, life is more prolonged, and in one instance, recorded by Dr. Hare,[1] the patient attained the age of thirty-eight years. Again, all clinical signs of the condition may be absent throughout a long life, if it should be confined to one side, the opposite kidney being healthy, and if the sac should not attain any considerable size. Under such circumstances the hydronephrosis may be only discovered at post-mortem examination. In acquired cases the phenomena characteristic of the accumulation may be preceded by those due to its cause, more especially by symptoms of renal colic resulting from the passage of a calculus along the ureter, which either lodges there permanently, or sets up morbid changes in its walls which lead to the ultimate closure of the duct. The special signs of hydronephrosis may be summed up as an enlargement or tumour in the renal region, increasing more or less rapidly, usually unaccompanied with any pain or tenderness, occasionally presenting a lobulated outline, and having a soft, elastic, or fluctuating feel; while the urine is usually normal or but slightly altered, and in some instances comes away in large quantity suddenly, with simultaneous subsidence of the tumour, which subsidence may be permanent, or occasionally this event occurs from time to time, the enlargement again forming in the intervals between the excessive discharges of urine.

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[1] Pathological Transactions, vol. xiii. p. 137.

When first discovered, a tumour which is due to hydronephrosis corresponds in its situation to other forms of renal enlargement, occupying the lumbar region, and reaching back to the spine; but as the accumulation increases, it extends more or less upwards, and downwards into the hypochondriac and iliac regions, as well as in a forward direction. The size which it ultimately attains varies considerably, and an enormous tumour may be formed, completely filling the abdomen, and leading to great general enlargement of this part of the body, so that the origin of the swelling cannot be recognized, and it has under such circumstances been mistaken for an ovarian cyst. As it increases, it sometimes can be felt to present a distinctly lobulated outline; while it usually yields to the finger a sensation more or less clearly indicative of an enclosed collection of fluid, namely, a feeling of softness, elasticity, or fluctuation, which a change of posture does not affect. A hydronephrotic tumour is not in itself in the least degree painful or tender as a rule, but it may be accompanied with pain due to its cause, such as a calculus in the ureter; and paroxysmal attacks of pain in the direction of this duct have been experienced in exceptional cases. More or less discomfort and uneasiness, or a sense of fulness or tension, may also be felt when the tumour becomes very large; and griping pains are occasionally induced in consequence of its interference with the intestines. Occasionally the swelling itself seems to be somewhat tender. When both kidneys are involved, of course there will be an enlargement on each side, but not usually to an equal degree. The percussion-sound is absolutely dull and toneless over the site of the tumour; but if it is not very large, the colon can generally be detected, by its special resonant sound, lying in front of it. Change of posture does not in the least influence the dulness.

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[1] Medical Times and Gazette, 1858.

The absence of any abnormal characters in the urine is an important fact in the clinical history of most cases of hydronephrosis, as, when only one kidney is involved, the opposite organ becomes hypertrophied, and forms an adequate quantity of healthy excretion. Occasionally it contains a small quantity of pus; and in connexion with paroxysms of nephritic colic blood may be present. The discharge of a profuse quantity of urine, which is usually somewhat modified in its characters and composition, with the coincident disappearance of an enlargement in the renal region, is highly characteristic of hydronephrosis, indicating that some obstruction has been removed, and thus the accumulated fluid has been able to escape suddenly. Of course such an event can only take place under certain conditions, and it may be repeated a variable number of times, the ureter becoming again blocked up, and the urine consequently retained, with subsequent relief of the obstruction.

A hydronephrotic sac interferes more or less with neighbouring structures, and thus may originate symptoms. The most frequent of these are due to impaired action of the intestines, there being constipation and flatulence, and complete obstruction of the bowels may be induced, or in other cases dysenteric symptoms may be excited. The stomach may also be affected, with consequent vomiting. In rare instances one or other of the chief veins is compressed; while the diaphragm and thoracic organs may be interfered with in extreme cases. The general health is also liable to be more or less affected.

The clinical course and ultimate issue in cases of hydronephrosis are variable. If the morbid condition is one-sided, should the obstruction be removed, and the retained urine escape, recovery may ensue, with partial or complete destruction of the affected kidney, the opposite organ assuming double duty, but it may subsequently become the seat of disease, and death may be the consequence. Very rarely does a hydronephrotic sac rupture internally and thus lead to a fatal result.

In some instances death has been preceded by uræmic symptoms, either in consequence of both kidneys being involved, or of a calculus obstructing the ureter leading from the healthy organ, or exceptionally even where one kidney was perfectly free and healthy. A fatal termination may also ensue from the immediate or remote effects of the removal by operation of the contents of a hydronephrotic sac. Peritonitis may be thus excited, or suppuration set up, with consequent wasting and hectic fever. The pressure-effects of the accumulation have in exceptional cases either been the actual cause of death, especially intestinal obstruction, or they have aided in bringing about this result.

Diagnosis.—Hydronephrosis cannot be diagnosed if the quantity of fluid accumulated is small; and it is only when the sac attains such dimensions as to give rise to a perceptible enlargement that the complaint can be recognized. With regard to its positive diagnosis, assistance may be derived from the past history, either revealing that the condition is congenital, or that there has been some cause likely to give rise to hydronephrosis, such as the passage of a calculus; and also indicating the clinical course of the case. The important characters of the enlargement are, that it usually occupies the ordinary site of a renal tumour, having the colon in front; that it is not painful or tender; and that it presents tactile sensations indicative of a fluid accumulation. The sudden discharge of a large quantity of urine, with subsidence of the tumour, is pathognomonic. Hydronephrosis may be mistaken for morbid conditions within the abdomen which are not connected with the kidney, where there is an accumulation of fluid, but especially for ascites or an ovarian cyst. This difficulty, however, should only be likely to happen if the sac attains a very large size, or if both kidneys are affected. Usually an ovarian tumour can be traced into the pelvis; it does not occupy the lumbar region, which is resonant, while it is entirely dull in front; and special modes of examination will reveal other characters pertaining to this disease. If a hydronephrotic sac becomes extremely large, it may be impossible to distinguish the two conditions, and cases have occurred in which tapping has been performed for a supposed ovarian cyst, which proved to be hydronephrosis. The diagnosis between this condition and ascites might present difficulties if it were double, but the fluid being confined does not move about with change of posture, as it does in ascites, and thus no alteration is observed in the shape of the abdomen, in the seat of fluctuation, or in the percussion-sound. The history of the case, and the accompanying clinical phenomena, will in cases of doubt aid in the diagnosis.

With regard to other affections of the kidneys which are attended with enlargement, hydronephrosis is in but little danger of being confounded with any solid growth; but it has to be distinguished from swelling due to other accumulations of fluid, namely, renal abscess, pyonephrosis, or perinephritis, from hydatid disease, and from a simple cyst in the kidney, which in rare instances attains a very large size. Suppuration within or outside the kidney is usually acute in its progress; the enlargement is painful and tender; there may be superficial signs of suppuration; and general symptoms indicative of this process are more or less prominent. In connexion with pyonephrosis pus is passed mixed with the urine, or there is a history of such admixture. The general system is also much affected, there being usually wasting, with hectic fever, and repeated rigors. Hydatid disease is almost always unilateral; other structures may be implicated at the same time; hydatid-fremitus may be elicited in some cases, or small hydatid cysts or echinococci may be passed in the urine. A simple cyst cannot be distinguished by any definite signs. The aspirateur may prove of much value in doubtful cases in the diagnosis between hydronephrosis and other morbid conditions.

PROGNOSIS.—This is somewhat uncertain, and differs much in different cases, but there is always more or less danger to life. The chief circumstances which influence the prognosis are whether the hydronephrosis is single or double, and when it is single, the condition of the opposite kidney; the amount of the accumulation, and its effects on surrounding structures; the possibility of any obstruction being removed; and the effects produced on the general system. The chief sources of danger arise from the supervention of uræmic symptoms, from interference with the intestines or other organs, or from rupture of the sac, the last event being, however, extremely rare. Cases of unilateral hydronephrosis may go on for an indefinite time, if the accumulation is not large, and a cure may result if any obstruction can be got rid of, or in some instances after the fluid has been removed by operation. After either mode of evacuation, however, the fluid may re-accumulate, and after an operation suppuration is liable to take place. Double hydronephrosis is necessarily very serious, and uræmic phenomena usually lead to a fatal termination, but it is sometimes remarkable what a length of time life may be prolonged even in cases of this kind.

TREATMENT.—The indications for the treatment of hydronephrosis are, first, to endeavour to get rid of any obstruction to the escape of urine, or to cause the urine to pass by such obstruction; or, secondly, failing this, to remove the accumulated fluid by operation. The first indication can only be carried out in certain cases, where the escape of urine is prevented by some morbid condition causing pressure upon the ureter, or by internal obstruction of this tube, as by a calculus; or where there is some congenital closure of the urethra, which can be remedied. When an impacted calculus has originated hydronephrosis, careful manipulation and shampooing over the enlargement may be practised, and this has been attended with success, either in the way of keeping the accumulation within moderate limits, or even of getting rid of it altogether. If this plan of treatment is inadmissible, then comes the question of operation. Should the enlargement be unilateral and of small size, while there are no prominent symptoms, it is certainly advisable not to interfere; but should it be of considerable dimensions, or progressively enlarging, thus becoming troublesome and evidently affecting adjoining structures injuriously, it is desirable to remove the fluid. This can generally be done with safety by means of the aspirateur, or it may be requisite to use a trochar and cannula, and it will be well always to adopt antiseptic precautions. Should the fluid re-accumulate it may be again removed, and repeated tapping has been resorted to with success in some instances. In cases of double hydronephrosis it may also be advisable to operate, but no definite rule can be laid down. If only one kidney is involved, care must always be taken to try to preserve the opposite organ in a state of health, by regulating the diet, and maintaining a free flow of watery urine. The general system may require attention in cases of hydronephrosis; as well as any symptoms or complications to which this condition may give rise.

RENAL ABSCESS.

BY FREDERICK T. ROBERTS, M.D., B.SC., F.R.C.P.

DEFINITION.—It is convenient to regard any accumulation of pus within the kidney as a renal abscess, but this may originate in different ways, as was first clearly pointed out by Rayer. Thus, it may arise from suppuration in the substance of the kidney, following inflammation—*suppurative nephritis;* from pyelitis, with subsequent closure of the ureter, so that the pus collects within the renal pelvis—*pyonephrosis;* or from a combination of these conditions—*pyelo-nephritis.* From either of these causes the whole kidney structure may be ultimately destroyed, nothing being left but a sac containing pus, which is often mixed with other materials. Peri-nephritic or peri-renal abscess results from inflammation affecting the capsule and the tissues which immediately surround the kidney—*peri-nephritis;* the pus therefore accummulating around instead of within this organ. The formation of these abscesses is generally attended with prominent local and general symptoms, and they usually tend to open in certain directions, thus giving rise to more or less serious consequences.

SYNONYMS.—Suppurative Inflammation of the Kidney; Nephritis Vera; Acute Interstitial Nephritis. Pyæmic abscesses have been separated by some writers from suppurative nephritis, their synonyms being metastatic or multiple abscesses.

ÆTIOLOGY.—The formation of one or more abscesses in the substance of the kidney may be due to the following causes:—1. Direct injury over the renal region, by which the kidney structure is wounded or contused, as from a stab, gun-shot, blow, kick, or fall. 2. Irritation of the substance of the organ, especially by one or more calculi lodged there, by which inflammation may be excited, terminating in suppuration. Here may also be mentioned the fact that hydatid or other cysts in the kidney occasionally undergo inflammatory changes, ending in the formation of an abscess; and that morbid growths occasionally excite suppurative inflammation, or may even break down into a kind of abscess, as in the case of tubercle. 3. Suppuration in the renal pelvis, ureter, bladder, or urethra, to which the same process in the kidney is secondary, being originated either by direct extension, through the agency of the blood, or in some other way not definitely determined (*see* Consecutive Nephritis). 4. Embolism, with consequent hæmorrhagic infarctions, which may ultimately become converted into abscesses, involving also the renal tissues. 5. Pyæmia, in the course of which disease the kidneys are liable to become the seat of abscesses, though much less commonly than some other organs. In this class of cases the suppurative process is also probably due to small embola of a septic nature. It may follow pyæmia from any cause, but is especially common after suppuration of the urinary passages, though, on the other hand, the kidneys may be free when disease in these passages is the cause of pyæmia. 6. Extension from surrounding structures or neighbouring organs in very rare instances.

Pyonephrosis is usually preceded by chronic pyelitis, which leads to the production of pus, and this accumulates along with the urine in the renal pelvis, just in the same way as urine alone collects in cases of hydronephrosis, in consequence of some obstruction preventing its escape through the ureter. The pyelitis generally owes its origin to calculous disease, and it is the impaction of a stone which is the most frequent cause of the obstruction. The affection may, however, be due to retained and stagnant urine which has become decomposed, or to irritation by parasites or by morbid products, such as cancer or tubercle, and either of these may block up the ureter, or this effect may be due to a blood-clot, inspissated pus, or mere inflammatory swelling or ulceration.

Perinephritis and its consequences most commonly follow suppuration in connexion with the kidney or its pelvis, the inflammatory process being set up either by mere extension, or from the bursting of an accumulation of pus within the kidney into the surrounding structures. Rokitansky says that it is particularly liable to supervene whenever a tedious inflammatory process is kept up by calculous disease; and in such cases one or more calculi may also find their way into the peri-renal tissues, and thus increase the irritation. The escape of urinary fluid from the kidney into these tissues may likewise set up perinephritis. More or less inflammatory changes around the kidney are likely to be excited by morbid growths in this organ, especially carcinoma, but these are of a more chronic nature, and lead to the formation of adhesions. Occasionally perinephritis originates from some external violence inflicted over the renal region; and it has in rare instances been regarded as idiopathic, resulting from a cold. Niemeyer[1] states that he had seen one case in which a peri-cystitis extended along the ureter to the fat around the kidney.

Anatomical Characters.—Suppuration in the substance of the kidney is usually preceded by marked congestion, either uniform or scattered here and there throughout its structure. Hence the organ is of a deep red colour, enlarged and softened, while a quantity of bloody fluid escapes on section. It then becomes infiltrated with exudation, the tubules are filled with epithelium, nuclei, and granules, and the interstitial tissue presents abundant leucocytes; consequently the colour alters, becoming whitish or yellowish, and suppuration afterwards takes place in isolated spots, which may extend and coalesce, until finally a large portion or even the whole of the kidney is destroyed, being converted into a bag of pus, which may attain a large size. Abscesses in pyæmic cases are usually separate, of small size, and scattered in greater or less number in the midst of the renal structures, both kidneys being involved. Other forms of renal abscess are generally limited to one organ. The collection of pus may open into the renal pelvis, and be discharged by the ureter, the remains of the kidney subsequently shrinking; or it may burst in certain other directions, to be presently indicated. In exceptional instances the contents of an abscess become inspissated and caseous, or even calcareous, a cure being thus effected.

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[1] Text-book of Practical Medicine, vol. ii. p. 40.

Pyonephrosis is preceded by chronic pyelitis, and hence the lining membrane of the pelvis and infundibulum presents marked changes. It is altered in colour, being paler than normal, and often of a grey or slate-colour; while enlarged veins are frequently visible upon it. It is also thickened, and either unusually tough and firm, or softened. The epithelium is destroyed, erosions are common, and in some instances actual ulceration is observed. Deposits of exudation, caseous matter, or other morbid products upon its surface are not uncommon. The pelvis, infundibulum, and calices become distended to a variable degree, and the renal tissue is then involved in different ways, until finally only remnants of it are left, or it may even be destroyed altogether. The organ may be merely compressed by the accumulated fluid, and becomes gradually wasted, as already described in connexion with hydro-nephrosis. Or it is implicated in the suppurative process, either by direct extension, or in isolated spots in its substance—*pyo-nephrosis;* and thus undergoes disintegration and destruction. In the condition last-mentioned it is affirmed that abscesses seated deeply in the cortical substance, and apparently shut in, can be traced to communicate with the pelvis by a tract of suppuration extending along the tubules to the apices of the pyramids, showing their origin in extension of suppuration along these tubules. According to the extent of the destructive processes just indicated will the morbid appearances vary. Thus there may be a sac of variable size, corresponding to the dilated pelvis, containing pus and other products, with scattered abscesses throughout the kidney; or the organ may have entirely or almost entirely disappeared, nothing remaining but a sacculated space, which may ultimately attain a considerable size. Even should any renal tissue be left undestroyed, it will probably be found to have undergone morbid changes of a chronic inflammatory nature.

The contents of the cavity in cases of pyo-nephrosis consist chiefly of pus, usually mixed with retained urine. The latter tends to decompose, and consequently the fluid becomes ammoniacal and alkaline in reaction, while the pus is found to have undergone more or less alterations from its healthy characters. The quantity of fluid thus accumulated differs much in different cases, and it may be very considerable. Mixed with it are not uncommonly found other materials, such as blood, calculi or their *débris*, portions of morbid growths, precipitates of urates or phosphates, or, in exceptional instances, scales of cholesterin. In course of time deposits are likely to form upon the inner surface of the sac, usually consisting of secondary phosphates. The purulent fluid may also become gradually inspissated by the precipitation of calcareous and magnesian phosphates and carbonates, and this may lead to a practical cure, the contents becoming more or less putty-like or chalky, and the walls of the cavity contracting upon them. These walls sometimes become calcified or even imperfectly ossified, as well as the septa which partially divide the interior of the sac. In other instances the pus escapes through the ureter, in consequence of the removal of the obstruction, the sac then shrivelling up, or it may alternately fill and be emptied for a variable number of times. The accumulation of pus may also open in certain directions.

In perinephritic abscess pus is found around the kidney, suppuration taking place in the surrounding structures, which are chiefly composed of adipose tissue. This is preceded by the ordinary signs of inflammation. According to the cause of the affection, the abscess may or may not be found to communicate with the interior of the kidney; and when such a communication exists, urine, calculi, or other materials which have escaped from this organ, may be found mixed with the pus. The quantity of matter formed is often very considerable. A perinephritic abscess tends to burst in certain directions, if allowed to take its course, and as this termination may also happen in the case of purulent accumulations within the kidney, it will be convenient to indicate here the directions in which such opening may occur. In the first place the abscess may make its way to the surface, and burst externally, either in the lumbar region, in the iliac fossa, at the upper part of the thigh, the matter passing beneath Poupart's ligament along the psoas magnus muscle, or even in the gluteal region. Occasionally there is more than one external opening. Or it may rupture internally, communicating either with the colon or, on the right side, with the duodenum; with the peritoneum; or, rarely, with the pleura or lung, after perforating the diaphragm. Pathological consequences are likely to arise from these modes of termination, such as peritonitis or pleurisy; and when the abscess bursts externally, sinuses with fistulous openings are liable to remain. In a case which has long been under the observation of the writer, two such sinuses exist, which are constantly discharging, the one opening in the lumbar region, the other in the iliac fossa.

In cases where suppuration takes place in connexion with the kidney, especially if chronic, it is not uncommon to find other parts of the urinary apparatus involved, to which, as already pointed out, the renal condition is usually secondary. For instance, there may be cystitis, inflammation and suppuration of the ureter, or suppuration in the urethra. Moreover, if there be a chronic discharge of pus from one kidney, the opposite organ is liable to become the seat of albuminoid or other forms of disease; and other organs may be similarly affected. Pulmonary phthisis may also be set up.

SYMPTOMS.—The formation of abscesses in the kidney itself may occur without any definite symptoms pointing to implication of this organ. This is especially liable to happen in cases of pyæmia, or when the suppuration is secondary to a similar process in the urinary passages or bladder. In other instances symptoms connected with the kidney are present, but none characteristic of this disease; and it may happen that an abscess which has not been previously recognized bursts into the renal pelvis, its existence being only indicated by an unexpected discharge of purulent urine. Usually the complaint is acute, and the phenomena which may be anticipated in its course, *local* and *general*, are of the following nature.

Pain is experienced over one renal region, which often attains considerable intensity, is liable to exacerbations, and shoots downwards towards the hypogastrium, scrotum, or upper part of the thigh. It is much aggravated by movement, and also by pressure, there being in some instances extreme tenderness over the loin. When suppuration takes place, the pain may become of a throbbing character. Retraction of the testicle is sometimes observed. Micturition may be too frequent; while the urine is generally much affected, both as to its amount and characters. This excretion is diminished in quantity, and may be almost suppressed. That which is passed is of a deep colour, concentrated, and frequently contains an admixture of albumen or blood; but neither hæmaturia nor albuminuria is constant. As suppuration progresses, should an abscess attain a sufficient size, it affords external objective signs of its presence. A fulness or tumour forms in the region of the kidney, which comes to yield a sensation of more or less elasticity or fluctuation. Ultimately, should the abscess tend to open externally, the superficial structures present signs of inflammation, and pointing may be observed. The purulent accumulation may, however, burst into the pelvis of the kidney, and this event is indicated by a copious discharge of pus with the urine, and a coincident diminution or disappearance of the swelling. The purulent discharge may soon cease, or it may continue as a chronic condition. The clinical signs of the evacuation of a renal abscess in other directions will be presently indicated. It must be remembered that even after such an abscess has formed, and its existence has been recognized, it may undergo a curative process without opening in any direction.

Acute suppurative nephritis is usually ushered in by rigors, and these frequently continue at more or less regular intervals during the progress of the disease, especially when pus has formed. There is marked pyrexia, with its concomitant phenomena: and nausea and vomiting are often prominent symptoms. The fever tends to assume a low type at an early period, and in many cases the symptoms become of a marked typhoid character, with a dry brown tongue, sordes on the lips and teeth, and low nervous phenomena, such as mental wandering, muttering delirium, a tendency to stupor or coma, tremors or twitchings, or convulsive movements. These phenomena are due partly to the suppurative process, partly to the interference with the renal functions and consequent blood-poisoning. When they occur, they usually lead to a fatal termination.

It has already been remarked that pyæmic abscesses in the kidneys do not generally give rise to any symptoms attracting attention to these organs. Such abscesses may, however, be suspected if, in a case of pyæmia, there is evident tenderness over the lumbar regions, while the urine is much diminished in quantity, and contains blood or a considerable amount of albumen. If suppuration follows disease in the bladder or urinary passages, the progress is more chronic, and the general symptoms assume a more or less hectic type.

Pyonephrosis and its consequences are usually, but not invariably, preceded by symptoms of pyelitis, and especially by a discharge of purulent urine, which has lasted a variable time. When the obstruction occurs which prevents the escape of the pus and urine from the pelvis of the kidney, the pain and suffering become aggravated, especially if they are due to a calculus, micturition becomes more frequent, and provided the pyelitis is unilateral, the urine which is discharged ceases to contain pus and is clear, because it comes from the healthy organ. At the same time a fulness or tumour forms in one renal region, which increases more or less rapidly, is painful and tender, and comes to present a sensation of softness and elasticity or fluctuation. This fulness is usually confined to the loin, or occupies the space between the margin of the thorax and the iliac crest; it may, however, extend across the middle line of the abdomen. Should the obstruction be got rid of, the fulness subsides, with coincident appearance of abundance of matter in the urine. The outlet may again become blocked, the tumour forming anew, and again it may subside, owing to the passage once more becoming free. This filling and emptying of the pelvis of the kidney may alternate for a number of times. In some cases of pyonephrosis the sac ruptures either internally or externally, or it is emptied by operation; in exceptional instances it remains as a chronic condition, the patient gradually sinking from its effects, and in others the contents become by degrees inspissated and dried up, a cure ultimately resulting. It is impossible to determine clinically in cases of pyonephrosis, with any degree of certainty, when the kidney structure itself is involved in the suppurative process. There may be indications of the development of chronic Bright's Disease, in the occurrence of diminished

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The course of events which occurs after renal or peri-renal abscess opens externally or is evacuated by operation, varies in different cases. It may heal up immediately or gradually, no further accumulation taking place; or, on the other hand, the case ends fatally sooner or later. In other instances the matter collects again, and another opening is formed, perhaps in some other direction, or two more operations are required, which may lead to ultimate recovery, as happened in a case which came under the writer's observation. Again, one or more sinuses or fistulæ may remain, discharging pus, which may at the same time come away with the urine. The condition last-mentioned is accompanied in time with general wasting, fever, night-sweat, and a tendency to hectic; while phthisis or albuminoid disease is liable to be set up, if the discharge continues for any length of time. By judicious management, however, the general health may even in these cases be maintained in a fair condition for a long period, and secondary lesions warded off.

DIAGNOSIS.—It will only be practicable to touch briefly upon the principal points relating to the diagnosis of suppuration within or around the kidney. In the first place, it must be remembered that under certain circumstances renal abscess may be entirely latent, and cannot be recognized during life, especially in cases of pyæmia. In other instances the diagnosis presents little or no difficulty, the nature of the disease being clearly indicated by the history and course of symptoms. Neither pyo-nephrosis nor perinephritic abscess is ever likely to be clinically latent, each of these conditions being usually indicated by prominent local and general signs.

Occasionally a difficulty is experienced in distinguishing a collection of pus within or around the kidney from neighbouring morbid conditions. The chief of these to be borne in mind are abscess or hydatid disease of the liver or spleen; localized accumulation of fluid in the peritoneum; and suppuration in the superficial structures, depending upon bone-disease or other causes. The diagnosis must be determined by a careful consideration of the history of the case; the precise situation and limits of any enlargement; and the presence or absence of renal symptoms. It may happen, however, that a large accumulation of pus forms within the pelvis of the kidney, without the occurrence of any urinary symptoms, as in a case recorded by Caffe,[1] where a supposed cyst in the spleen, which was operated upon by Nelaton, proved to be pyonephrosis. Again, when a renal or peri-renal abscess, in making its way to the surface, passes into some unusual locality, it may be difficult to trace it to its source, and to distinguish it from other abscesses. Sinuses and fistulæ remaining after the collections of pus have opened externally, may also present obscurity as to their origin. In one such case which came under the writer's observation, the simultaneous discharge of purulent urine proved that sinuses opening externally had originated from suppuration within the kidney, and probably from pyonephrosis.

The next point bearing upon the diagnosis is to distinguish suppurative diseases from other affections of the kidney which cause enlargement in connexion with this organ. There is not likely to be any difficulty with regard to solid growths, such as cancer, though these do present in some instances a sensation of quasi-fluctuation which might mislead; the chief conditions, however, which must be borne in mind are fluid accumulations associated with the kidney, namely, hydronephrosis, cystic disease, and hydatids, which yield to palpation sensations resembling those afforded by a collection of pus. Such a collection is, however, distinguished from the conditions just mentioned by the history of the case; the presence of pain and tenderness in the renal region, usually accompanied by prominent urinary symptoms and marked alterations in the urine, as well as with general symptoms indicative of suppuration; and frequently by the more acute progress of the case. Hydatid cysts may yield hydatid fremitus; other organs are usually similarly affected; and vesicles may be passed with the urine. It must not be forgotten that these cysts may ultimately suppurate; while hydronephrosis, owing to decomposition of the retained urine, occasionally terminates in pyonephrosis.

A collection of pus in connexion with the kidney being recognized, it then becomes necessary to determine whether it is due to renal suppuration, pyo-nephrosis, or peri-nephritis; and, if possible, to trace the morbid condition to its cause, whether injury, calculus, tubercular disease, &c. These conclusions can only be arrived at by a careful consideration of each case in all its details, past and present, and much difficulty may be experienced, while it must be borne in mind that the conditions may be more or less combined. As distinguishing pyonephrosis from abscess in the kidney, the former is preceded by symptoms of pyelitis, perhaps for a considerable time; there may be clinical indications of some obstruction causing the retention of pus, with coincident clearing-up of the urine; and alternate filling-up and evacuation of the renal pelvis may take place, with corresponding changes in the characters of the urine, which is very characteristic. The diagnosis of perinephritic abscess is founded upon the superficialness of the pain and tenderness, with more marked exacerbation on movement; the signs of superficial inflammation with subcutaneous œdema; and the absence of any marked changes in the urine, though it must not be forgotten that this excretion is sometimes much altered, even when there is no communication with the kidney.

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[1] Gaz. des Hôp. 1855.

The implication of the kidney or its pelvis secondarily, as a consequence of previous disease in the bladder or urinary passages, may be very difficult to ascertain. The development of local pain and tenderness in the renal region may call attention to this complication, especially if accompanied with the escape of a considerable amount of pus with the urine, and with more marked general symptoms than were previously observed, namely, fever, wasting, and hectic. It may happen that a more or less fluctuating fulness forms over the region of the kidney, and this will establish the diagnosis.

The last point relating to the diagnosis is the recognition of the bursting of an accumulation of pus associated with the kidney into the various internal structures, and the conditions resulting therefrom. If it is known that such an accumulation exists, the phenomena already described as characteristic of the different modes of termination usually sufficiently indicate what has happened, particularly if any enlargement over the region of the kidney subsides simultaneously.

PROGNOSIS.—The formation of a collection of purulent fluid within the kidney, whatever be its cause, is an event necessarily attended with much danger, and in a considerable proportion of cases leads, sooner or later, to a fatal issue. This may result either from interference with the renal functions and its consequences; from acute general symptoms of a typhoid character, due to the suppuration; from the bursting of the accumulation into internal parts; from gradual wasting and hectic in connexion with the chronic formation and discharge of pus; or from secondary lesions set up in consequence of long-continued suppuration. Recovery may take place, however, either after the purulent collection has been evacuated spontaneously, or by operative interference; or as the result of inspissation or calcification of the pus, and shrinking of the sac containing it, so that no further mischief results, although the kidney is more or less destroyed, the opposite organ enlarging and performing extra work.

The prognosis will be influenced by various circumstances, among which the following demand special notice:—In the first place, the cause of the renal affection is important. For example, if it is associated with pyæmia the prognosis is necessarily extremely grave, although the fatal termination then results rather from the general condition than from the local lesion. Again, when suppuration in the kidney or its pelvis arises as a complication of disease of the bladder or urinary passages, or when pyonephrosis follows tubercular or cancerous pyelitis, the case is exceedingly unfavourable. When the cause is of a merely local nature, such as injury or calculous disease, of any urinary symptoms, as in a case recorded by Caffe,[1] where a supposed cyst in the spleen, which was operated upon by Nelaton, proved to be pyonephrosis. Again, when a renal or peri-renal abscess, in making its way to the surface, passes into some unusual locality, it may be difficult to trace it to its source, and to distinguish it from other abscesses. Sinuses and fistulæ remaining after the collections of pus have opened externally, may also present obscurity as to their origin. In one such case which came under the writer's observation, the simultaneous discharge of purulent urine proved that sinuses opening externally had originated from suppuration within the kidney, and probably from pyonephrosis.

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[1] Gaz. des Hôp. 1855.

If suppuration in the kidney takes place slowly, no active measures can be adopted to prevent it. Should pus accumulate in the renal pelvis owing to some obstruction preventing its escape, this might possibly be got rid of by careful shampooing and manipulation along the ureter, as described under hydronephrosis. If this result could be effected, treatment directed to the pyelitis would then be indicated, so as to endeavour, by means of astringents and other remedies, to check the formation of matter, at the same time the cause of the disease being got rid of, if possible. Should the kidney-affection be developed secondarily to disease in other portions of the urinary apparatus, of course this primary disease must receive due attention, especially if connected with the bladder or urethra.

Sooner or later it happens in a good many cases that a collection of pus in connexion with the kidney attains such a size as to justify or demand its removal by operation. The time when this has to be resorted to must depend on individual judgment, but it must be remembered that there is always a danger of the abscess opening into some internal part, and also of the kidney being entirely destroyed, and therefore unnecessary delay ought to be avoided. From his own experience, the writer would certainly recommend that, whatever the cause of the accumulation of pus might be, whether renal abscess, pyonephrosis, or perinephritis, the matter should first be removed by means of the aspirateur, and the operation might be repeated even more than once if the fluid should again collect. Antiseptic precautions should be duly employed. In a case of supposed perinephritis which came under the writer's observation, aspiration was performed on two occasions, and the patient then made a good recovery. He died from some other affection some years afterwards, and at the post-mortem examination no trace of any suppuration could be discovered, but the kidney was wasted and shrivelled to merely a small remnant. If aspiration does not succeed, then it is requisite to make a free opening, and especially if there are signs of superficial suppuration. This should usually be made in the posterior lumbar region, and the patient the prognosis is more hopeful. It is also affected by the amount of the purulent accumulation, and is much worse if both kidneys are involved. Further, the course of events will materially modify the prognosis. Suppose the case to be one of pyo-nephrosis, the removal of the obstruction preventing the escape of the pus and urine will be a favourable sign; and even if there be alternate filling and emptying of the sac for a number of times, recovery may ultimately ensue, though with destruction of the kidney. If a renal abscess makes its way towards the surface and opens externally, especially in the loin, a cure may also result, though most cases which terminate in this way succumb sooner or later. Undoubtedly judicious treatment, having for its object the removal of the fluid by operative interference, will materially affect the prognosis under these circumstances, and may lead to a favourable issue which could not otherwise be expected. The bursting of any collection of pus into internal parts is always a very serious event. If the communication should form with the peritoneum or thoracic cavity a speedily fatal termination may be anticipated; if with the bowel, the prognosis is a little more hopeful, though it is only in very exceptional cases that recovery takes place, the progress towards death, however, being usually slow and gradual.

With regard to perinephritic abscess, much that has been stated above applies also to this morbid condition. If the kidney is healthy, and the perinephritis is not secondary to suppuration within the organ, the prognosis is decidedly more favourable than under opposite circumstances. The chief dangers arise from the abscess bursting inwardly, in the different directions already indicated; and from the general symptoms which are associated with it, either acute or chronic.

In all these conditions, the previous state of the patient will to some extent influence the prognosis; and it must also be guided by the evident effects upon the constitution which they induce, especially when they terminate in a chronic discharge of pus, some individuals being much less affected by this event than others.

TREATMENT.—If symptoms point to the occurrence of acute inflammation, either affecting the kidney itself or its surrounding tissues, which is likely to lead to suppuration, the local removal of blood from the corresponding lumbar region is decidedly indicated, provided the condition of the patient admits of this measure. It is in traumatic cases that it would most probably be required, while of course it is not to be thought of in pyæmia, and is seldom admissible if there has been previous disease of the urinary apparatus. Blood may be removed by means of leeches or cupping, the quantity taken away being determined by the state of the patient. Cupping may be performed to the extent of from four to twelve ounces; or from half-a-dozen to a dozen leeches may be applied. Free dry-cupping may be practised when blood cannot be abstracted. The persistent application of cold has been advocated, by means of the ice-bag or of

TUMOURS AND NEW GROWTHS OF THE KIDNEY.

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DEFINITION.—The expression "renal tumour," or "tumour of the kidney," is used in a somewhat indefinite sense, and is generally made to include several pathological conditions which are entirely distinct from each other in their nature and origin. Thus it is applied to any form of enlargement, which depends either upon a morbid state of the kidney itself, upon some fluid accumulation within its pelvis, or upon perinephritis and its consequences. It therefore comprehends conditions which have already been described in previous articles, in addition to certain new formations or growths in connexion with the renal apparatus. It must be mentioned, however, that such new formations by no means necessarily cause enlargement of the kidney, at any rate to such a degree as to render the organ recognizable as a tumour during life. Nevertheless it will be expedient to discuss in the present article all morbid conditions coming under this category, which are not elsewhere considered.

GENERAL SUMMARY.—The diseases belonging to the class of new formations which it is proposed now to consider, are indicated in the following table:—

I. Cystic formations.	1. Simple cysts. 2. Congenital cystic degeneration. 3. Cystic degeneration in adults.
II. Malignant growths.	Encephaloid, Scirrhus, Colloid, Epithelioma, Melanosis, &c.
III. Non-malignant growths.	Sarcoma (?), Fibroma, Lipoma, Myxoma, Glioma, Angioma. Adenoma, &c.
IV. Tubercular disease.	Nephro-phthisis.
V. Syphilitic disease.	
VI. Parasitic formations.	1. Hydatids—Echinococcus hominis. 2. Cysticercus cellulosus. 3. Strongylus gigas. 4. Bilharzia hæmatobia, &c.

Before entering upon the separate discussion of these morbid conditions, it will be convenient to point out the general characters of a *renal tumour*, by which it can usually be distinguished from other abdominal enlargements:—1. The situation of the tumour is of much importance. In the first instance it occupies more or less of the lumbar region, in the interval between the margin of the ribs and the iliac crest. It is often more apparent to inspection and palpation towards the front or lateral portion of this region than posteriorly, as there is more resistance in the last direction, but the fingers cannot be passed behind the swelling, so as to feel its hinder border. Manipulation will show that the enlargement does not extend into the pelvis; and it can also usually be ascertained by palpation or percussion that it does not pass upwards within the margin of the thorax, though it may reach more or less into the hypogastrium. In a forward direction it may also encroach upon the abdominal cavity to a variable extent. 2. Palpation may reveal that the outline of the tumour corresponds more or less distinctly to the shape of the kidney. Its lower and anterior limits can generally be felt, and it is rounded on these aspects. The surface is often smooth, but may be irregular; while the consistence differs according to the cause of the enlargement, though it is usually firm. 3. The tumour is generally completely fixed, and its position can never be influenced to any appreciable extent by manipulation, or by the diaphragmatic movements accompanying deep breathing. 4. Percussion gives important information. Over the greater part of the enlargement there is absolute dulness, which extends posteriorly to the spine. In front, however, a line of tympanitic resonance can be usually elicited, in consequence of the colon lying in front of the kidney, and more or less of the small intestines may also occupy this position, and thus influence the percussion-sound. On the right side, even if a renal tumour cannot be separated from the liver by palpation, percussion may yield an intestinal note at its upper end, and thus the two organs can be distinguished. The sensation felt by the finger on percussion varies according to the cause of the enlargement, being frequently, however, that of firmness and resistance. Elasticity, fluctuation, or hydatid-fremitus may be felt in certain conditions.

It must be noted that renal enlargements are liable to deviate considerably from the general description just given, and it is necessary to be prepared at the outset for such deviations. For example, an accumulation of fluid or a solid growth may attain such dimensions as to pass altogether beyond the limits of a renal tumour, sometimes indeed completely filling the abdominal cavity, so that, except by the history of its seat of origin and progress, it will be impossible to determine its connexion with the kidney. Sometimes a growth is limited to one end of the organ, and thus seems to be connected with some adjacent structure. Again, a renal tumour occasionally presents pulsation and a bruit. One important mode of examination in doubtful cases consists in the use of the aspirateur or exploratory trochar, which may clear up any difficulty experienced in determining whether an enlargement is of renal origin, as well as in making out its exact nature.

Having given this general outline, the several morbid conditions will now be considered in the order in which they are enumerated in the table given above.

I. Cystic Formations.

1. *Simple Cysts.*—These are of not infrequent occurrence in kidneys which are otherwise perfectly healthy, especially in elderly subjects, and they are commonly found in these organs when they are the seat of the granular contracted form of Bright's Disease.

With regard to their pathology, it is a well-established fact that these cysts originate mainly in dilatation of portions of the urinary tubules, but some of those formed in Bright's Disease are due to distension of the Malpighian capsules. Why dilatation of the tubules should take place in healthy kidneys is not very clear, but it has been attributed to blocking-up of their channels by fibrinous particles. The development of cysts in the granular kidney is more evident, for the ducts are obviously obstructed at intervals, either by plugs of exudation, or as the result of pressure upon their exterior by contracting fibrous tissue. Consequently the intermediate portions become distended. Two other views have been advanced to account for the origin of these cysts. Simon attributed them to great enlargement and development of the epithelial cells lining the urinary tubules; while it has also been maintained that they are developed out of the intertubular connective tissue. Neither of these theories has, however, gained much acceptance. In the rare cases in which a cyst in the kidney has attained large dimensions, there has been an obscure history of external injury.

The cysts which are observed in otherwise healthy kidneys are usually superficial, often projecting somewhat above the surface, and when the capsule is removed, their contents may escape. Some may be situated more deeply, but they are chiefly confined to the cortical portion. They vary in number and size; and may be scattered or grouped. Generally they range in size from a pea to a nut or walnut, but may be very minute, or, on the other hand, a cyst in rare instances reaches large dimensions, so that it occupies a considerable portion of the abdominal cavity, and contains a large quantity of fluid. The walls are very thin and delicate, and indeed the superficial cysts sometimes appear to have no structure between them and the capsule of the kidney. The contents are usually fluid, clear, transparent, and of a yellowish colour. This fluid is not of the nature of urine, for it very rarely contains urea, though more frequently a small amount of uric acid is found dissolved in it. It is generally albuminous, and contains carbonates and phosphates, as well as in some instances a good proportion of cholesterine. A jelly-like or colloid substance is also commonly found in these cysts, varying in consistence from a thin gelatinous fluid to an almost solid material. This may completely fill the smallest cysts, but usually floats in the fluid as irregular masses. In a case recorded by Dr. Hare,[1] the contents of a large cyst were fluid when first removed, but set into a tremulous jelly in a few minutes after exposure to the air.

The cysts observed in the granular kidney are more numerous than those just considered, being often in large numbers, and always of small size, many of them being very minute, while they rarely become larger than a pea. They are scattered chiefly through the cortical substance, and may be so abundant as to make this appear as if it were not atrophied. In the pyramidal portion they are sometimes elongated, and present a moniliform appearance, being placed end to end. Their walls are thick and firm, and a delicate lining of epithelium may be perceptible. In cysts which originate from Malpighian corpuscles, the remains of the vascular tufts may be discovered. The contents are similar to those in the cysts already described.

These simple cysts do not give rise to any clinical signs of their presence, unless one of them should attain such dimensions as to form a perceptible tumour, which only happens in extremely exceptional instances. Such a tumour may, however, be of a considerable size, compressing and interfering with surrounding structures more or less. It is painless, and presents a sensation of softness or fluctuation. The condition is very liable to be mistaken for hydronephrosis, and perhaps also for hydatid disease. The past history and the use of the aspirateur might afford some assistance in diagnosis. Of course when the cysts are associated with the granular kidney, there will be the usual symptoms characteristic of this disease.

2. *Congenital Cystic Degeneration.*—This is a very remarkable morbid condition, but it is more of pathological than of clinical interest. Its mode of development is doubtful. Virchow and Förster have shown that the cysts have their origin in the urinary tubules and Malpighian bodies, as is the case with the simple cysts already described. Virchow maintains that the cause is to be found in inflammation of the straight ducts in the pyramidal portion of the kidney, occurring in the fœtus in utero, an embryonic nephritis. Consequently the walls of these ducts adhere, and their channels become closed up. The result is that the urine cannot escape from the kidney, and its tubules become dilated at intervals, as well as the Malpighian capsules. The minute pouches thus formed enlarge and become developed into cysts, being at the same time separated from each other, and the intervening portions of the tubes become obliterated. Virchow further holds that the inflammation depends upon the impaction of crystals of uric acid and urates in the ducts. As a consequence the renal papillæ waste, and the pelvis becomes obliterated. Koster has started another view as to the pathology of congenital cystic disease, namely, that it is due to

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[1] Pathological Transactions, vol. iv. p. 199.

a primary want of development of the apparatus for carrying away the urine, analogous to atresia ani and other malformations of this kind. In favour of this theory he advances the fact that the calices, pelves, and ureters are absent in many instances, but this is not always observed. The fact that other congenital malformations affecting the limbs and other parts exist in some cases, also seems to support this view. It is not improbable that both theories are correct in different instances.

Congenital cystic degeneration of the kidneys almost always involves both organs. They are usually more or less enlarged, and may equal in size adult organs. Specimens have been found to measure as much as six inches in length, and four inches in width and thickness. In a case described by Dr. Duffey,[1] each kidney weighed six ounces. On the other hand, in exceptional instances the organs are much diminished in size, and shrivelled. Their surface is generally irregular, owing to the projection of cysts, but it may be quite smooth. On section the kidney is found to be converted to a variable degree into a mass of cysts, which are often in great numbers, and of different sizes. Usually some remnants of renal structure are observed between the cysts, but this may have disappeared entirely. Generally also there is more or less increase in the connective tissue, but not always. The cysts are lined with epithelium. Their contents vary, the smaller ones containing a fluid holding urinary ingredients in solution, the larger ones an albuminous fluid; a colloid substance may also be found in some cysts. Occasionally the kidneys present a spongy or cavernous appearance on section, and the cysts are only visible on microscopic examination.

Associated with congenital cystic degeneration of the kidneys, there is in the large majority of cases found to be some malformation, variable in its nature, involving other parts of the urinary aparatus, namely, either of the calices and pelves, the ureters, the bladder, or the urethra. Other portions of the body may also give evidence of imperfect development as indicated by cleft palate, absence or malformation of a limb, anencephalia, and allied conditions.

It mostly happens that those who are the subject of this disease are born prematurely, or, if the fœtus attains the full period, it is not viable, in consequence of the mechanical interference with the descent of the diaphragm caused by the enlarged kidneys, so that respiration cannot be initiated or carried on. In some cases embryotomy has had to be performed before delivery could be accomplished, in consequence of the large size of the abdomen of the fœtus. If the morbid condition has not proceeded far, the child may be born alive. It is a curious fact that cystic degeneration is sometimes observed in several children in succession, or alternately with healthy children, the mother being also quite healthy.

3. *Cystic Degeneration in Adults.*—This is a much rarer condition than the congenital disease. It probably originates in the same way,

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[1] Dublin Quarterly Journal, vol. xli. p. 438.

that is, from dilatation of the urinary tubules at intervals, and also of the Malpighian bodies. It is a question whether this affection is not merely a further development of a slight degree of the congenital form, which has gradually increased after birth. This may be the case in some instances: but in others the morbid condition seems to have commenced during extra-uterine life, and it has then been attributed to inflammation of the straight tubes in the pyramids, with consequent obliteration at intervals; or to plugging of these tubes by small coagula of blood. It has been regarded as being related to the small granular kidney. This disease has never been observed under thirty years of age, and most of the patients were between forty and fifty; it has been met with even above sixty. Men suffer twice as often as women. Its commencement has occasionally been apparently connected with injury over the renal region, and consequent hæmorrhage into the kidney.

Cystic degeneration of the kidneys in adults always involves both organs, but usually unequally. They are enlarged and increased in weight, often enormously. In a case recorded by Dr. Hare[1] the left kidney measured 15¼ inches in length, 9½ in breadth, and about 23 in circumference, and weighed 16 lbs. The right was only enlarged to about double its natural size. Bright[2] has described a case in which the left kidney was eight or ten times, and the right at least six times the natural size. In two cases brought forward by Dr. Whipham[3] the combined weights of both kidneys were respectively 81 ounces and 81¾ ounces. When the morbid change is advanced, the surface is always more or less irregular or lobulated, owing to the projection of cysts, and the kidney may have entirely lost its normal outline. On being cut into, it is seen to consist of an immense number of closed cysts, differing much in size, usually ranging from a pea to an apple, but some cysts may contain as much as a pint of fluid. The smaller cysts sometimes project into the larger ones, and they may communicate, owing to absorption of their contiguous walls. There is generally some renal tissue left, except in extreme cases; and the connective tissue is much increased in quantity. The contents of the cysts are usually liquid, clear, and yellowish or straw-coloured. They may be red, purple, or chocolate-coloured, from the presence of blood or altered blood. Sometimes they are opaque, inspissated, and semi-solid; or they may be of a gelatinous nature. The fluid is always albuminous, but does not contain urinous elements. Under the microscope, blood-corpuscles or their *débris*, cells from the renal tubules, pus-corpuscles, oil-globules and granules, crystals of triple phosphates, or uric acid, or abundant plates of cholesterine have been observed in different cases. The walls of the cysts are more or less thick according to their size; and they are lined by epithelium. Occasionally a cyst suppurates, and bursts into the renal pelvis. The urinary passages and bladder are perfectly free and open, and as a rule they are quite healthy.

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[1] Pathological Transactions, vol. iii. p. 131.
[2] Abdominal Tumours, New Syd. Soc. Publications, vol. vi. p. 208.
[3] Pathological Transactions, vol. xxi. p. 244.

Clinically cystic degeneration of the kidneys is not revealed by any characteristic phenomena during its earlier stages, its progress being very chronic and insidious. The disease may prove rapidly fatal from uræmia, when there have been no previous symptoms pointing to the kidneys. It would always be difficult to determine its existence with certainty during life; still, several cases have been diagnosed correctly, and the affection might be indicated by signs sufficiently characteristic. The condition as it advances necessarily interferes seriously with the renal functions, and especially with the excretion of the solid urinary ingredients. Occasionally pain is complained of in the loins, which may assume the character of recurrent and violent paroxysms. The urine does not seem to be diminished much in quantity, if at all, even until a late period, and it may be considerably in excess; but it is usually watery, and of low specific gravity. Albuminuria and recurrent hæmaturia are common, but not essential symptoms. The development of a renal tumour constitutes an important feature in the clinical history. This may be observed only on one side, and it may attain a large size, reaching even beyond the middle line; but most frequently an enlargement can be detected on both sides, and this may assist the diagnosis. The tumour may have an elongated form, and usually feels soft, but not fluctuating. Sometimes it presents a firm sensation, either over its whole extent or in parts. Patients suffering from cystic degeneration are often very emaciated and weak; sometimes œdema of the legs is observed. They are frequently troubled with dyspeptic and intestinal disturbances; while vomiting is sometimes a prominent symptom. Death is generally preceded by uræmic symptoms, such as mental confusion and delirium, coma, twitchings or convulsions, epileptiform fits, general paralysis, or violent hiccough. This event may, however, result from independent complications, such as bronchitis, which the renal condition will necessarily render more dangerous.

II. MALIGNANT GROWTHS—CARCINOMA—CANCER.

ETIOLOGY AND PATHOLOGY.—Cancer of the kidney is a comparatively rare form of disease, though not so uncommon as was formerly supposed. It may be either primary or secondary, and in the former case other structures are often subsequently involved. When it is primary, the cause of the development of the affection is generally quite undiscoverable, and it must be confessed that we are ignorant as to its origin. In some instances malignant disease has been attributed to injury over the loin, or to some direct irritation of the kidney, mechanical or chemical; but this is very doubtful, for even when there is a history of such an event, it may only accidentally have drawn attention to a previously existing condition, or if it be looked upon as the immediate cause of the cancer, there is no explanation why the disease should arise in one person and not in another. This can only be referred to a special constitutional state, but at the same time it is only in a small proportion of cases of primary malignant disease of the kidney that any hereditary predisposition has been traced. Even in the case of young children there is no family history of cancer, and other children of the same family are quite healthy. Age exercises a remarkable influence as regards the frequency of this affection. Formerly it was regarded, like other forms of cancer, as a disease almost confined to persons advanced in years. More recent observations have, however, revealed the fact that a large proportion of cases are met with among very young children. Of sixty-seven cases collected by Dr. William Roberts,[1] twenty-five occurred in children under ten years of age, of whom twenty-two were under five years. Dr. Braidwood[2] describes four cases which came under his notice in children under two years of age, in one instance the disease having been first observed when the child was four months old, death occurring at eight months. One of these cases the writer had an opportunity of seeing during life. The disease is very rarely met with from ten to twenty years of age, but is not proportionately uncommon in young adults, though it becomes more frequent from fifty to seventy. Males suffer much more frequently than females from renal cancer; but this difference is observed to a far greater degree in adults than in children.

Secondary cancer of the kidney may result from direct extension from some neighbouring structure; or as a development of a general carcinomatous diathesis, this organ being implicated along with, but at a subsequent period to others, which have been the primary seat of the disease.

With regard to the pathology of true renal cancer, it is not yet satisfactorily settled from what tissues the elements which constitute the growth originate. It was formerly generally supposed, and is still held by some pathologists, that they were developed from the cellular tissue of the kidney. Braidwood believes that the cancerous alteration commences in the cellular tissue surrounding the Malpighian corpuscles. More recently, however, the cancer cells have been traced to the epithelium lining the urinary tubules, from the proliferation of which they are supposed to be derived. The stroma consists of the walls of the tubules and the intertubular connective tissue, which afterwards becomes increased in some cases. It must be noticed, however, that in some instances, which are apparently those of renal cancer, it is probable that the disease has not really commenced in the actual substance of the kidney, but in its hilus, spreading thence into the organ. Wilks,[3] indeed, thinks that this is the commonest plan of attack in primary renal cancer, and observes: "It is remarkable how seldom the proper tissue of the kidney can be said to be primarily affected; for although a large tumour may exist, and this after death be removed as renal cancer, yet a careful dissection discovers the disease to have arisen primarily in the lymphatic glands, or other parts outside the kidney, especially about its hilus, while the organ itself is comparatively untouched; the disease, however, puts on the form of the kidney, for it has probably penetrated the hilus and expanded the capsule." Zencker and Karl Schroeder have also drawn attention to a similar mode of origin in cases of supposed primary renal cancer, the growth being termed "paranephritic," and the latter observer believes that such growths originate in the endothelium of the blood-vessels. It is highly probable that this view of the origin of renal cancer is correct in many instances.

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[1] On Urinary and Renal Diseases, 3rd edit. p. 525.
[2] Liverpool Med. and Surg. Reports, vol. iv. p. 45.
[3] Pathological Anatomy, 2nd edit. p. 518.

ANATOMICAL CHARACTERS.—Primary renal cancer is one-sided in the great majority of cases, but there is a difference of statement as to which organ is most frequently attacked. Out of sixty-seven instances collected by Dr. William Roberts,[1] sixty were unilateral cases, and each kidney was affected an equal number of times. In fifty-nine cases collected by Ebstein,[2] thirty-one involved the right, twenty-three the left, and five both kidneys. Encephaloid or medullary cancer is the variety met with in all but exceptional instances, though Waldeyer has designated many cases as simple carcinoma, and ranks them as intermediate between scirrhus and encephaloid. Well-marked scirrhus is very rare; occasionally masses have been found in the midst of medullary cancer. Colloid cancer has been observed in exceptional instances; or portions of other malignant growths have undergone colloid degeneration. Robin has given a description of one case of supposed epithelioma. Klebs has described growths presenting transitions from cancer to adenoma; and other tumours of a mixed character have also been described, consisting of carcinoma, sarcoma, and adenoma. Melanotic growths have been observed in the kidney, but it is doubtful whether most of these were of a cancerous nature. Faggé[3] has, however, recorded a case in which such growths were found in the kidneys along with other organs, and spots of pigment in the lining membrane of the pelvis. The same observer[4] has brought forward an instance of what he believed to be carcinoma lipomatosum, which occupied the whole centre of the right kidney, and grew freely into the renal vein and its branches. This growth presented the characteristic alveolar structure of a carcinoma, and the fat-globules were contained in the interior of cells of very irregular forms, with large oval nuclei.

For practical purposes it will be sufficient to describe the anatomical characters which cancer of the kidney ordinarily presents, and it may certainly be regarded as belonging more or less distinctly to the encephaloid variety. The morbid growth is either infiltrated uniformly throughout the organ, or assumes a nodular form. The kidney is almost always enlarged to a variable degree, and increased in weight. It may assume enormous dimensions, and reach several pounds in weight, often growing with great rapidity; and it is remarkable that the largest and heaviest specimens have been observed in children. Dr. William Roberts[1] found that in the cases collected by him where information was given as to the weight, in children it averaged 8½ lbs., the smallest being 1 lb. 9 ozs., the largest 31 lbs.; in adults the average weight was 9½ lbs., but in one case the size and weight were about normal, and the highest weight recorded was 27 lbs. The shape of the kidney depends on the mode in which the cancer is distributed in the organ. If the growth is infiltrated, the outline may be but little or not at all altered, only that the organ seems more rounded than usual; if it is nodular, then the kidney becomes more or less irregular in form, and presents prominences of various sizes upon its surface, giving it a lobulated appearance. Sometimes the growth projects from one point of the organ, and then the irregularity is still more evident. The colour is frequently altered from that of the normal kidney. The consistence varies, and is often unequal; some of the nodules may feel firm, but usually they are more or less soft, and sometimes present a sensation of quasi-fluctuation.

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[1] On Urinary and Renal Diseases, 3rd edit. p. 524.

[2] Ziemssen's Cyclopædia of Medicine, vol. xv. p. 662.

[3] Pathological Transactions, vol. xxviii. p. 173.

[4] Ibid. vol. xxvii. p. 204.

A section of a cancerous kidney presents different appearances according to the extent to which the change has advanced, the mode of arrangement of the growth, the variety to which it belongs, its degree of vascularity, and the alterations it may have undergone, and to the condition of the renal tissue, should any remain. The cortical portion is first implicated, and then the disease extends into the medullary portion. If it is in the infiltrated form, the whole section may present a uniform yellowish or whitish appearance. When the arrangement is nodular, nodules presenting this colour may be seen in the midst of the kidney-structure, from which they are sometimes separated by distinct capsules of condensed cellular tissue. The uninvolved renal-tissues may be healthy, congested, atrophied and the seat of degeneration, or inflamed and even suppurating. In many cases, however, the separate growths have coalesced, and have thus entirely destroyed these tissues. Again, they may present different degrees of vascularity, in some instances resembling the so-called "fungus hæmatodes;" while the numerous delicate vessels are liable to give way, being also sometimes the seat of minute aneurisms, so that extravasations of blood not uncommonly occur, which may be very considerable, as much as a pint of blood having been found in a cavity in the midst of a cancerous kidney. Moreover, the appearance and characters of the cancerous tissue may be varied by the occurrence of softening, fatty degeneration, the formation of cysts, or of abscess-like cavities. The consistence is said generally to be about that of human brain, but this is also variable, and the material may be quite pulpy.

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[1] Urinary and Renal Disease, 3rd edit. p. 523.

Most commonly cancer of the kidney projects into the pelvis of this organ, and its walls may be involved. Blood is also liable to be extravasated here, and the clot may assume a stratified arrangement. The pelvis is sometimes distended or distorted. The ureter is often occupied more or less by an extension of the cancerous growth, and its channel may be thus completely closed, or its walls may be implicated. This tube is also liable to be obstructed by blood-clots, or as the result of pressure upon it by the renal tumour. Calculi are found sometimes in the pelvis of a cancerous kidney. The renal vein is frequently involved in the disease, being first surrounded and compressed, then its walls are destroyed, and the growth gradually fills its channel, and may thence spread directly into the vena cava inferior, or even beyond this vessel.

A cancerous kidney usually remains in its normal position, and becomes united to surrounding structures by firm and extensive adhesions, its capsule being also thickened and often highly vascular. Occasionally the organ is displaced, and it may be free and movable. As it enlarges it produces various effects upon neighbouring organs, either displacing, compressing, or perforating and destroying them, or they become involved by extension of the morbid process. The colon always lies in front of the growth, but is often compressed, and may be quite flattened and collapsed. Among the conditions which have been observed in exceptional instances are compression of the duodenum, with consequent dilatation of the stomach; perforation of the duodenum; erosion of the vertebræ; and perforation of the abdominal wall. The vena cava inferior is liable to be pressed upon more or less, and anasarca of the lower extremities results; ascites is only rarely observed. When the growth becomes of considerable size, it interferes with the diaphragm, pushing it upwards, along with the thoracic organs.

In more than half the cases of primary renal cancer, secondary formations have been observed. The structures most commonly thus implicated are the absorbent glands in the hilus of the kidney, those lying on the vertebræ, and the mesenteric glands. Very large tumours may be found in connexion with these structures. Next in order come the lungs and liver, but other organs or structures are much less frequently involved. It is a remarkable fact that the bladder and lower urinary passages are extremely rarely affected secondarily in cases of cancer of the kidney.

In unilateral renal cancer, the unaffected organ is generally hypertrophied, and thus does double duty; in rare instances it becomes the seat of amyloid degeneration.

Secondary cancer is not uncommon in the kidneys, and when the condition is a manifestation of a general cachexia, both organs are usually involved. The carcinoma exists in the form of scattered nodules, which may range from not larger than a pin's head to the size of a walnut. The organs are not enlarged, and their shape is not altered. The intervening renal tissue is quite healthy.

SYMPTOMATOLOGY.—Renal cancer may be latent throughout its course, giving rise to no symptoms or signs whatever. This is observed in almost all cases of secondary cancer, which is generally only discovered at the post-mortem examination, but only very rarely happens when the disease is primary. The local clinical phenomena to which it will be necessary to call attention as more or less significant of cancer of the kidney, are pain in the renal region, hæmaturia, and the development of a tumour. In addition to these, there are frequently symptoms due to the interference by the growth with neighbouring structures; and others connected with the general system.

Pain is an uncertain symptom in cases of renal cancer. Its absence or insignificant character has been often noted throughout or for a considerable time, even when the tumour has attained a large size. On the other hand, marked pain may be complained of from an early period, and although its absence by no means excludes cancer of the kidney, its presence along with a tumour and hæmaturia is of importance in making a positive diagnosis. It may be experienced, too, before the other more characteristic signs of the disease are observed, and may thus be the means of drawing attention to this serious condition, by leading to a careful investigation of the case. The pain is referred mainly to the lumbar and hypochondriac regions on one side, but is liable to shoot along the lower intercostal spaces, or downwards towards the inner side of the thigh or along its posterior aspect, in the latter case resembling sciatica, and there is then a danger of the suffering being attributed to mere neuralgia, should there be no other prominent symptoms. In character the pain may be constant, dull, and aching, or intermittent, and it is often aggravated paroxysmally, sometimes becoming intense at intervals. There is no retraction of the testis. Tenderness is usually observed when there is spontaneous pain in connexion with a tumour, and in some cases the pain is only felt on manipulation or pressure over the renal region, when it may also sometimes be caused to shoot into other parts. When a cancerous kidney assumes large dimensions, its mere bulk will probably give rise to sensations of more or less discomfort, pressure, or tension. It must also be borne in mind that attacks of true renal colic may occur in cases of this disease, owing to the passage either of calculi or of blood-clots from the pelvis of the kidney to the bladder.

Hæmaturia is a symptom of much importance in many cases of cancer of the kidney, but it was absent in rather less than half of the recorded cases, and it may only be observed at the outset of disease, subsequently disappearing, or towards its close. This is accounted for either by the fact that no hæmorrhage has taken place; or that the blood has been prevented from escaping, in consequence of the obstruction of the ureter by a clot or by cancerous matter, or of its compression by the tumour. Occasionally also it is due to some other cause than the carcinoma, such as a calculus. In not a few instances hæmaturia has for some time been the only symptom of malignant disease, and exceptionally there has been no other symptom throughout; if it should occur spontaneously, without any pain or other symptom of calculus, the blood evidently coming from the kidney, this disease should be suspected and watched for. Sometimes the first appearance of blood in the urine is traceable to some external violence over the loin; and this has even drawn attention to the existence of a tumour in this region. The chief characters of hæmaturia occurring in connexion with renal cancer, which may be regarded as more or less significant, are, that it comes on spontaneously and without any obvious cause; that it takes place at irregular intervals, lasting a variable time; that the blood is in considerable quantity; and that there is no pain accompanying its discharge. The urine almost always affords distinct evidence of the presence of blood, and the discharge is not uncommonly very profuse, though rarely so excessive as to lead to general anæmia and faintness. The blood is mostly intimately mixed with the urine, but sometimes clots form, and these, during their passage, are liable to give rise to certain disturbances, especially to symptoms of renal colic during their transit along the ureter, when they may also interfere with the further escape of blood, the urine becoming quite clear; and to irritability of the bladder, accompanied with retention of urine and much suffering, from the impaction of a clot in the neck of the bladder or in the urethra. When examined microscopically the urine presents blood-corpuscles, some of which may be altered in appearance, and often blood-casts are visible.

The development of a tumour is the most frequent clinical phenomenon in cases of primary renal cancer, a distinct enlargement, readily discovered on examination, having been observed in all but exceptional instances. It first appears in the lumbar region, in the interval between the margin of the thorax and the iliac crest, but is usually more evident towards the front of this region than towards the back, because the growth meets with more resistance in the latter direction. The tumour increases sometimes with great rapidity, especially in children, extending chiefly forwards into the umbilical region, and downwards into the iliac region and towards the pubis. The size it may ultimately attain varies much, but it sometimes completely fills the abdomen, causing great general enlargement of this part of the body, particularly in children. When the growth becomes large, the superficial veins over it may be distended. When it has not exceeded moderate limits, palpation reveals that it has rounded margins, and its surface is either smooth or irregularly nodulated and lobulated. The consistence is variable, but generally the sensation afforded is one of more or less elasticity, and sometimes of quasi-fluctuation; not uncommonly different parts of the tumour yield sensations of different degrees of firmness, especially when it is lobulated. A cancerous kidney is almost always remarkably immovable, on account of the adhesions which form, so that it is not affected by the deepest inspiration, or by manipulation. At the same time, when the tumour projects anteriorly, and its connexion with the kidney is not very evident, firm pressure upon it with one hand from the front in a backward direction, while the other hand is placed behind, may push it back somewhat, and show that it belongs to this organ. In exceptional instances also the tumour is not fixed, and a floating kidney may become the seat of cancer.

Percussion yields a dull sound over the greater part of the tumour in cases of renal cancer, but in front an intestinal note is usually elicited, owing to the colon lying in front of it, and on the left side not uncommonly a portion of the small intestines occupies the same position. Sometimes the colon is compressed, however, and then there is dulness also in front; but the intestine may then possibly be felt as a band lying upon the growth. It has been suggested that in this case the gut might be blown up by injecting air per rectum. The opposite side of the abdomen is generally tympanitic, in consequence of the mass of the small intestines being pushed over by the tumour, provided it has not become so large as to fill the abdominal cavity. A sensation of more or less resistance is experienced on percussion. In exceptional instances renal cancer has distinctly pulsated; and still more rarely a blowing murmur has been heard on auscultation. Holmes[1] has recorded a case in which both pulsation and a bruit were present.

The urine, apart from the presence of blood in it, does not present any special changes in cases of renal cancer. Sometimes the quantity is increased or diminished, and the nitrogenous elements may be in excess. Albuminuria and pyuria have been occasionally observed, depending, however, upon other conditions than the malignant disease. There has been much discussion as to whether cancer-elements are ever found in the urine. The balance of evidence is certainly against the detection of any microscopic elements characteristic of cancer, and cells which have been supposed to have come from such a growth, have probably been merely epithelium particles detached from the renal pelvis and ureter.

The symptoms which may result in cases of renal cancer from the interference of the tumour with neighbouring structures are variable. The bowels may be much constipated or even obstructed; occasionally there is diarrhœa. Gastric symptoms are not uncommon, such as loss of appetite, dyspepsia, nausea, or vomiting, but the appetite may be retained to the last, and is sometimes excessive. Local œdema or anasarca of the legs, with enlargement of superficial veins, may be observed as a consequence of pressure on large veins. In rare instances jaundice has occurred from obstruction of the common bile-duct.

The general system is usually gravely affected, as evidenced by rapid emaciation, which may become extreme, progressive debility and exhaustion, anæmia, constitutional irritation and restlessness, and a yellowish tint of skin. In some instances, however, general symptoms have not appeared for a considerable time. The pulse is sometimes unusually slow, and the temperature below the normal.

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[1] Pathological Transactions, vol. xxiv. p. 149.

Febrile complications occasionally occur towards the end. Symptoms may arise indicating secondary development of cancer in other organs.

The duration of cases of renal cancer varies widely, but it is shorter in children than in adults. Dr. William Roberts[1] found that in children the average duration was nearly seven months, the extremes being ten weeks to "over a year"; in adults the average was two-and-a-half years, the extremes five months to seven years. Death usually results from gradual exhaustion, but it has also taken place from rupture of the cancerous kidney. Uræmic symptoms have never been observed in cases of this disease.

III. NON-MALIGNANT OR BENIGN GROWTHS.

NON-MALIGNANT growths are only very rarely found in the kidney, and they are of but little clinical significance. A brief account of these growths will therefore suffice.

1. Sarcomata have been observed in several instances, but usually as secondary formations, being then evidently malignant. In rare cases a primary growth is of this nature, presenting on microscopic examination a spindle-celled or round-celled structure. It is doubtful whether such a growth should be regarded as malignant or benign. It is usually of small size, but may attain such dimensions as to be recognizable as a tumour during life.

2. Fibromata are generally observed in the form of small nodules or knots, situated near or in the pyramidal portion, either in kidneys which are otherwise healthy, or in those which are granular and contracted. They do not give rise to any symptoms. They are very dense, and are composed of firm fibrous tissue, sometimes enclosing the remains of renal tubules. Wilks[2] has recorded a case in which the right kidney was converted into a tumour of the size of a young child's head. This consisted of a large fibrous growth, which had invaded and destroyed the whole structure; and of a cyst, which was the distended pelvis. The general form of the kidney was preserved. The new growth was remarkably hard, and resembled a fibro-cartilaginous tumour in appearance; but microscopic examination showed that it consisted entirely of fibrous tissue. Its centre was breaking up, so that a rugged cavity existed in the middle. In this case hæmaturia and pain in the loins had existed for ten years, and a tumour formed six years before death, which gradually increased. The health did not suffer much, and death was preceded by symptoms of a uræmic character. The patient was a male, aged fifty-three.

3. Lipomata or fatty growths are sometimes found in connexion with the kidney. They seem to commence generally around the organ, and to extend into its interior, affecting its nutrition, and lead-

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[1] Urinary and Renal Diseases, 3rd edit. p. 530.

[2] Pathological Transactions, vol. xx. p. 224.

ing to atrophy of its tissues. Ultimately a large portion, or even the whole, of the kidney may be converted into a mass of fat. Probably this condition in some instances follows atrophy of the kidney, and it has been found associated with calculi. Cases have been recorded in which tumours of large size were composed chiefly of fat in a matrix of fibrous tissue. Allusion has already been made under Cancer to a fatty growth observed by Fagge, and regarded by him as being of a malignant nature.

4. Without entering into any description, it will be sufficient to indicate what other forms of growth have been occasionally found in the kidney. Adenoid or lymphatic deposits are sometimes seen in certain cases of leucocythæmia and allied disorders. This tissue may even form actively growing tumours. Dr. Whipham[1] has recorded a case in which the kidneys were the seat of lymphadenoma, their surface being everywhere studded with small elevations, situated immediately beneath the capsule, and resembling to the naked eye extravasations of blood. Microscopic examination revealed that they were of a lymphadenomatous nature. Villous growths have been very rarely met with in the kidneys. Murchison[2] has recorded a case in which the mucous lining of the pelvis and calices of both organs presented numerous long villous processes, associated with a similar condition in the bladder. De Morgan[3] also described a case of a distinct villous tumour projecting from the wall of the pelvis of the left kidney. Part of the growth was compact, and consisted of a delicate fibrous stroma, from which a juice containing nucleated cells exuded. He regarded it as non-malignant. Other villi sprang from various parts of the walls of the pelvis. These growths may give rise to considerable hæmorrhage. Cavernous angioma is now and then observed in the kidney, usually near the surface. Gliomata occur as small tumours in the cortical portion, which may reach the size of a cherry, are very soft and tender, white and translucent, and deficient in blood. Lastly, cartilaginous and osseous tumours have been found in the kidneys, the latter resulting from the ossification of fibrous or other growths. Occasionally the capsule becomes ossified, forming a kind of shell.

IV. TUBERCULAR DISEASE—NEPHRO-PHTHISIS.

ETIOLOGY AND PATHOLOGY.—Cases which have been regarded as belonging to the class of tubercular diseases of the kidneys are referable to three groups, which it is necessary to distinguish, namely—1. Those in which so-called tubercle is formed primarily in the kidney or its appendages, and gives rise to prominent urinary symptoms. 2. Those in which the formation of tubercle is a manifestation of acute tuberculosis, the urinary organs being implicated along with other structures. 3. Those in which the disease occurs as a secondary event in the course of pulmonary or other form of phthisis, the renal condition being of the nature of a complication.

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[1] Pathological Transactions, vol. xxiii. p. 166.

[2] Pathological Transactions, vol. xxi. p. 210.

[3] Ibid. p 239.

The primary form of the affection is by no means common. The writer has only met with two cases which were apparently of this nature, during an experience of several years among the out-patients at the Brompton Hospital for Consumption. The true pathology of the disease is anything but settled. By many authorities it is regarded as a primitive tuberculosis—a manifestation of a constitutional condition—hence termed *primary renal phthisis, scrofulous* or *strumous inflammation*, and as analogous to the tubercular form of pulmonary phthisis. Other pathologists, with whom the writer is to some extent inclined to agree, are of opinion that the disease originates in inflammation, at any rate in many cases, the products undergoing a caseous or cheesy degeneration, but true tubercle often forms secondarily. Unquestionably in many instances there is no history of any hereditary tendency to phthisis. The affection has been in some cases attributed to cold, but this mode of causation is very doubtful, and it must be granted that generally the cause cannot be traced. The morbid condition in the kidneys is in some instances really secondary, and not actually primary, following cheesy or caseous inflammation either in the bladder, the testicle, epididymis, or the prostate gland; and it may originate by direct extension, or as a distinct and separate lesion, being then probably due to an infective process. It is remarkable that while the generative organs are frequently involved along with the kidneys in males, this is very rarely the case in females. Males are nearly twice as often affected with renal phthisis as females; in both the cases observed by the writer the patients were males. The disease is most common about the middle period of life, but has been noticed as early as three and a half, and as late as seventy-one years of age.

The etiology of secondary tubercular disease of the kidneys needs but little comment. As already indicated, it may be a mere local development of a general constitutional diathesis; or it arises as a complication of some previously-existing complaint, either in the lungs or elsewhere, which is usually recognized as being of a phthisical nature.

ANATOMICAL CHARACTERS.—In primary renal phthisis it is usually found that not only is the kidney and its pelvis involved, but almost invariably the disease also implicates the ureter, in a large proportion of cases the bladder, and sometimes the urethra. Originally probably only one organ is affected, but as the case progresses the other organ is often attacked, so that in the majority of instances examined post-mortem both kidneys have been found diseased, though usually to an unequal degree. The kidney itself may be first affected; or its pelvis, the lesion then spreading upwards to the kidney and downwards along the ureter; or the bladder or generative apparatus; or, occasionally, all the parts seem to be implicated almost at the same time, or in rapid succession. As a rule the morbid changes are seen in an advanced stage, but it will be requisite to point out briefly the course through which they pass.

When the disease starts in the body of the kidney, it is generally stated to begin in the cortical substance, and subsequently to extend into the pyramids; but Wilks thinks that the pyramids are earliest affected. At the outset grey nodules are said to form, but these rapidly become yellow and caseous, also coalescing to form extensive infiltrations, until finally the greater part, or occasionally even the whole of the organ is involved, its tissues being destroyed. Around these infiltrations miliary tubercles often appear. The cheesy masses soften in the centre, and come to resemble abscesses, which burst and discharge their contents into the renal pelvis. Thus irregular cavities, vomicæ, or ulcers are formed, having soft and cheesy walls, and from which a purulent fluid is discharged, mixed with caseous *débris*. Ultimately the entire organ may be destroyed in this way. In the pelvis and ureter the morbid formation commences in the sub-mucous tissue, as an irregular granular layer, semi-transparent at first, but soon becoming opaque and caseous. This also softens, and the overlying mucous membrane becoming thickened and ultimately destroyed, the disintegrated materials are removed, and irregular ulcers of variable extent are formed in the pelvis—the so-called *tubercular ulcers*—from which pus, blood, cheesy *débris*, and shreds of tissues escape. After a time other parts of the mucous membrane become thickened, discoloured, or covered with scrofulous deposits. This is the condition known as *tubercular* or *strumous pyelitis*. In rare instances thick calcareous deposits form. In some cases the ureter becomes almost completely closed up, either on account of infiltration of its walls, so that it forms a rigid tube, or from blocking-up of its canal by caseous masses or *débris*. Under these circumstances the escape of the pus and other materials from the renal pelvis is prevented, and pyonephrosis results. Then the kidney is involved, being either compressed by the accumulated fluids, or becoming the seat of the changes already described.

The exact appearances presented by kidneys and their appendages which are the seat of renal phthisis, necessarily vary considerably. The affected organ is commonly enlarged, either from the amount of the infiltration or from pyonephrosis; it may, however, be of normal size, or in exceptional cases is rather small. It is sometimes nodular at the surface; and its capsule is not uncommonly thickened and may be very firm, or it presents caseous patches upon its surface. Usually portions of the renal structure remain, which are either fairly healthy or in a state of disease. In extreme cases, however, the whole kidney is destroyed, nothing being left but a sacculated cavity, containing pus and other products. Occasionally these become inspissated, and the sac shrinks to a small size, a cure practically resulting under these circumstances. In rare instances the purulent collection has been known to burst in one or other of the directions already indicated in the article on Renal Abscess.

The bladder is frequently found to be the seat of tubercular disease, along with the renal apparatus, and in some cases the urethra is involved. The prostate gland, vesiculæ seminales, or testicles are also affected in a certain proportion of cases. The female generative organs are only very exceptionally implicated. In almost all cases secondary tuberculous disease will be found in some other organ or structure, especially in the lungs, in the intestines, or in the absorbent glands within the abdomen.

When tubercle in the kidney is a part of general tuberculosis, or is secondary to disease elsewhere, it assumes the form of small miliary granulations, either rounded or somewhat elongated, or linear if seated in the medullary portion, and often surrounded with a circle of excessive vascularity. These granulations form in the interstitial tissues, and consist of lymphoid cells. At first they are grey, but rapidly undergo caseation and become yellow. The granulations are chiefly observed towards the surface and in the cortical substance, and they are either scattered and distinct, or aggregated into groups. Both kidneys are almost always involved, but the pelves and ureters are not affected usually.

SYMPTOMATOLOGY.—When tubercle in the kidney is a part of general tuberculosis, or occurs as a complication of phthisis, it is only in exceptional cases that there are any symptoms present calling attention to this organ. When it arises in the course of phthisis, possibly the condition might be indicated by severe pains in the loins, with much tenderness, contraction of the lumbar muscles, and disordered renal functions, as in a case recorded by Colin.

Primary renal phthisis is usually characterized by prominent symptoms, but they may not appear for some time after the disease has commenced. It must be remembered also that the symptoms connected with the urinary organs are not uncommonly modified by the association of vesical or other mischief along with the renal affection. Pain in the lumbar region is present in the large majority of cases, and is not infrequently one of the first symptoms. It varies much in intensity, as well as in its characters, but seems in most instances to be dull and continuous, though it may occur in sharp paroxysms. It rarely shoots towards the thigh. Should the ureter become blocked, exacerbations of pain are liable to follow. Tenderness is also observed, and it may be present without any spontaneous pain. Micturition usually becomes very frequent, and there may be an almost constant desire to pass water, accompanied with straining, but little relief following the act, though this occasionally decidedly alleviates the pain and discomfort. Sometimes incontinence is observed. Pain or burning is often experienced during the passage of the urine. The disorders of micturition are generally more prominent when the bladder and urethra are involved, but these parts may be quite healthy, the symptoms being then traceable chiefly to the condition of the urine. This excretion presents striking characters, which are readily understood when the pathological changes observed in renal phthisis are borne in mind. It becomes almost always diminished in quantity as the disease progresses, though in exceptional cases it is in excess. The reaction remains faintly acid, provided that the bladder is not involved, or that the urine is not retained in the renal pelvis, when it tends to become alkaline and ammoniacal. Hæmaturia is usually observed at some period of the disease, but the blood is never passed in any considerable quantity. This may be one of the first symptoms noticed, and the blood may afterwards disappear. Small, thready clots are passed not uncommonly. One of the most striking characters presented by the urine is its admixture with pus, due chiefly to tuberculous pyelitis. At first the pus only causes slight turbidity, but afterwards it becomes abundant, so that it falls as a more or less copious deposit when the urine is allowed to stand. If the urine should become ammoniacal, the presence of the pus would cause it to become viscid and ropy, so much so that it might even be difficult to pass it. Albuminuria exists in proportion to the amount of pus discharged; and in rare instances this symptom has been observed very early, independently of the pressure of pus. Much stress has been laid, as significant of tubercular disease of the kidney, upon the presence in the urine of particles of caseous matter, which may be visible to the naked eye, and which are insoluble in acetic acid. Microscopic examination reveals in different cases and at different times blood-corpuscles; normal and altered pus-cells; epithelium cells from the urinary passages and bladder, often modified; granular detritus; and fibres of connective and elastic tissues, these being considered by some observers as highly significant.

Should the ureter become obstructed in any way, the clinical course of events will be similar to what has been described in former articles, namely, the clearing-up or diminished turbidity of the urine, and the development of a renal fulness or enlargement, which may exhibit fluctuation. This rarely attains any great size, and is often difficult to demonstrate; but it has been known to extend beyond the middle line of the abdomen. The fulness is generally painful and tender. It may vary in size and in the accompanying sensations from time to time.

Tubercular disease of the kidney is soon accompanied with general symptoms, which become more and more marked as the complaint progresses. These are pyrexia, often of a somewhat intermittent character, with regular chills or rigors, the fever in time assuming a hectic type, with night-sweats; general wasting, which may amount to extreme emaciation; anæmia; and debility. The digestive functions are also disturbed in most cases, and nausea and vomiting are not uncommon symptoms. As the disease advances, signs of implication of the lungs, intestines, or other organs are developed. Diarrhœa very frequently occurs. In rare instances a purulent accumulation may burst in some direction, and give rise to corresponding clinical phenomena. Possibly, if only one kidney is involved, a curative process may take place, and the patient recover. Almost always, however, the termination is fatal, and in most cases death has occurred within twelve months from the first appearance of symptoms, but the duration may be prolonged to two or three years. Death generally takes place from gradual exhaustion, aided by complications. If both kidneys are destroyed, uræmic phenomena may supervene at last. The rupture of a purulent accumulation may be the cause of the fatal termination in exceptional cases.

V. Syphilitic Disease.

The morbid condition of the kidney which is usually associated with syphilis is albuminoid disease, the connexion between the two being not infrequent. This lesion is discussed elsewhere, and need not be further considered here. The development of gummata in the kidney is exceedingly rare. Among the specimens exhibited at the Pathological Society during the discussion on Visceral Syphilis in 1877 (see Pathological Transactions, vol. xxviii.), there was only one of a gummatous growth in this organ. Even when present these gummata are usually very small, often not larger than a pea. The largest observed seems to have been in a case recorded by Dr. Moxon.[1] In the left kidney of a woman, which was the seat of albuminoid disease, a gummous tumour as large as a small potato was found. It presented a uniform, yellowish-white appearance, and was firm, tough, and dry. Microscopic examination showed that the growth consisted of small corpuscles crowded together, which gradually broke down into fat granules and globules, the renal tissue being destroyed and replaced. Other growths of this kind have presented similar characters. Ultimately they may be completely destroyed and removed, the kidney then presenting scar-like depressions.

The formation of syphilitic gummata in the kidney is not attended with any symptoms, and they have always been discovered at post-mortem examinations. Albuminoid disease occurring in connexion with syphilis presents the same clinical history as when it arises from other causes.

VI. Parasitic Formations.

The following so-called animal parasites have been found in connexion with the renal apparatus:—1. Hydatids, with Echinococcus hominis. 2. Cysticercus cellulosus. 3. Strongylus or Eustrongylus gigas. 4. Bilharzia hæmatobia. 5. Filaria sanguinis hominis, a minute nematoid worm, discovered by Dr. T. R. Lewis, associated with chylous urine. 6. Pentastoma denticulatum, a minute encysted parasite, only observed once, and supposed to be the larva of pentastoma tænioides. In addition to these, intestinal worms occasionally make their way into the urinary organs. Only the most important of these parasites are to be considered here.

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[1] Guy's Hospital Reports, 1868.

1. HYDATID DISEASE—ECHINOCOCCUS HOMINIS.

It is unnecessary to enter in this connexion into a general discussion of the natural history, etiology, and morbid anatomy of hydatid disease, as these matters are fully considered in the article on Hydatids of the Liver (Vol. iii.). It will be sufficient to draw attention to the main facts bearing upon the relation of this disease to the kidney.

ETIOLOGY.—The kidney stands third in the order of organs affected with hydatid disease, being much less frequently implicated than the liver, and also somewhat less commonly than the lungs. In exceptional cases other organs are involved at the same time; but usually the disease is confined to the kidneys. It occurs chiefly during middle life, but has been met with at the extremes of age. Males seem to be more often attacked than females in some places, probably because they are more exposed to the causes which lead to the introduction of the parasite into the system. In Iceland, however, this difference is not observed.

ANATOMICAL CHARACTERS.—Hydatid disease is almost always confined to one kidney, and the left is more frequently involved than the right. A cystic tumour is formed, which varies in size from that of an egg to that of an adult head, and it may project from the surface of the organ as a roundish prominence, elastic or fluctuating. Much will depend upon its situation. Usually the growth forms in the substance of the kidney, but sometimes it lies underneath the capsule, between this and the surface of the organ. When situated here or in the cortical substance, the cyst may attain a large size; if it occupies the pyramids, it usually bursts before it reaches any great dimensions. On examination the tumour is generally found to consist of the usual constituents of a hydatid growth, namely, an external fibrous capsule, lined by the laminated hydatid-cyst, this again being lined by a delicate germinal membrane, upon which scolices of echinococci are visible. The cyst contains a saline non-albuminous fluid, in which float secondary or daughter-cysts, and these in rare instances enclose tertiary cysts. Sometimes the structure is more simple, and there may be no secondary cysts or echinococci, this condition constituting the so-called acephalo-cyst. The fluid occasionally contains crystals of uric acid, oxalate of lime, and triple phosphates.

The effect of the hydatid-cyst upon the renal tissue is to cause it to waste more or less in proportion to the size of the tumour, and the kidney structure may ultimately entirely disappear, though usually some remains. The interstitial connective tissue is generally increased; and the growth often forms extensive adhesions with the parts around. The surrounding organs are more or less compressed and displaced if the cyst attains any considerable size.

The pathological sequence of events varies in different cases of hydatid disease of the kidney. The cyst may remain unchanged, or be opened artificially. In a very large proportion it ruptures into the renal pelvis, and especially if it occupies the pyramidal portion of the organ. Pyelitis may then be set up, and the small secondary cysts are conveyed away by the ureter. The cyst has also been known in exceptional instances to open into the intestines, stomach, or lungs and bronchi, in the last case its contents being expectorated, but never into the peritoneal cavity. It may open in more than one direction. The rupture may either occur spontaneously, or as the consequence of some external violence over the seat of the tumour. The cyst has occasionally suppurated, or has excited surrounding inflammation, terminating in the formation of an abscess, with which it has subsequently communicated. After it has ruptured, or now and then even without this event having taken place, it may become obsolescent or atrophied, its fluid contents being absorbed, and the sac shrivelling up into a more or less firm mass, consisting of a whitish, greasy, sebaceous-looking, or chalky material, which, on examination with the microscope, is found to be made up of fatty granules and globules, cholesterine, amorphous and crystalline phosphates, in which are embedded shreds of laminated membrane and hooklets of echinococci.

Occasionally renal calculus has been found associated with hydatid disease. In one recorded case one kidney was congenitally absent, and the other was the seat of a large calculus and of a hydatid-cyst. In the Pathological Transactions, vol. xxv., p. 174, an interesting case is described by Dr. Cayley and Dr. Woodman, of an infant in whom there was a multilocular cystic tumour, supposed to be a cystic form of hydronephrosis, combined with a true hydatid-cyst. Another case is also alluded to in the same communication where such a cyst was associated with encephaloid cancer.

SYMPTOMATOLOGY.—It need scarcely be remarked that hydatid disease of the kidney may be latent throughout, the condition being only discovered after death. In most cases no symptoms are observed for a considerable time, and the only phenomena which can be regarded as at all characteristic are, (1) the development of a renal tumour presenting certain characters; and (2) the passage of hydatid-vesicles or their structural elements, and their escape through the urethra with the urine. Various symptoms may arise from the events which are liable to occur in connexion with the tumour.

A tumour has been observed in about half the cases. It is almost invariably unilateral, and occupies the ordinary situation of a renal tumour, being as a rule confined within such limits that it can be recognized as belonging to the kidney. The growth is slow and gradual in its progress. Generally the tumour is painless and not tender; but if it becomes very large, it gives rise to a sense of discomfort, weight, or tension. On manipulation it feels smooth and rounded, and often tightly distended. Fluctuation is readily detected in some cases; in others it is indistinct, or there is only a sensation of elasticity. Hydatid-fremitus is a most important sign when present, and a sound of a similar character may occasionally be heard by auscultation on tapping gently over the tumour. Unfortunately, however, these signs are only elicited in a small proportion of cases, and they may be absent apparently even when the conditions exist which are regarded as most favourable for their production. Percussion usually reveals that the colon lies in front of the tumour, but not invariably.

The rupture of the hydatid-cyst, and the escape of its contents from the kidney, may happen either with or without the previous existence of an evident tumour, and it may occur spontaneously or from some obvious cause, such as a fall, a blow on the loin, or the jolting induced by riding or driving. It occasionally takes place once for all, the cyst becoming completely emptied, and subsequently shrinking; generally, however, this event is repeated at intervals varying from a few days to many years. The rupture may be preceded by pains in the renal region and hæmaturia. When it actually takes place, a sensation as if something had given way internally is frequently experienced by the patient, accompanied with much pain, and this is followed by symptoms more or less resembling those already described as characteristic of renal colic, these being due to the passage of the hydatid-vesicles along the ureter. Thus there are colicky pains, which may shoot down to the thigh or testicle; sometimes retraction of this organ; and, in severe cases, collapse, nausea, and hiccough. Should there have been a previous renal tumour, it might be expected that this should subside, but this is by no means always the case, as the ureter becomes blocked up, and hydronephrosis results. The symptoms last a variable time, and suddenly subside when the vesicle or vesicles reach the bladder, the hydronephrosis being then also relieved, if it should have been produced. After this, pains shooting to the end of the penis set in, with frequent attempts at micturition, strangury, and retention of urine. These symptoms are due to the transit of the vesicles along the urethra, and as soon as they are evacuated, immediate relief follows. They may be removed by the catheter, or, in the case of women, have been known even to be drawn out of the urethra by the fingers. Sometimes they are driven out by the stream of urine with great force. The pathognomonic sign of hydatid disease is the detection either of complete vesicles floating about in the urine, of collapsed vesicles, or of hooklets of echinococci and particles of laminated membrane. These broken elements form a whitish, milky detritus. The number of recognizable cysts discharged may vary from one to a great multitude. Blood is often present in the urine; and there may be a considerable amount of pus, owing to pyelitis being set up.

Hydatid tumour of the kidney sometimes occasions symptoms by interfering with other organs, and these may prove serious when the thoracic organs are impeded in their action. The opening of the cyst into the stomach, intestines, or bronchi can only be known by its elements being respectively vomited, discharged in the stools, or coughed up along with urinary constituents. If it should suppurate or set up suppurative inflammation around, this event will be accompanied with the local and general symptoms already described as indicative of renal abscess. Injury may be the cause of suppuration.

This affection is essentially chronic in its progress, often lasting for many years; but its duration is extremely variable and uncertain. In a large number of cases spontaneous recovery has taken place, usually after discharge of the vesicles. Death may occur from the rupture of the hydatid-cyst into the lungs; from thoracic complications set up by the tumour; from suppuration in or around it; from the effects of operation; or from independent maladies.

2. Strongylus or Eustrongylus Gigas.

This worm has been chiefly found in the kidneys and urinary passages of certain animals, such as the weasel, hare, ox, dog, wolf, &c., but is extremely rare in man. Only seven recorded cases have been regarded by Davaine as at all trustworthy. Strongylus belongs to the nematoids, and in general appearance resembles a very large lumbricus. The female measures from 14 to 25 inches in length, and the male from 10 to 22 inches; the breadth is a quarter of an inch or more. In addition to its great size, the strongylus is also characterized by its reddish colour, which is attributed to the sanguineous fluid in which it lies; and by having six papillæ or nodules around the mouth. It is quite unknown how this parasite reaches the kidney. It occupies almost exclusively the renal pelvis and calices, but has been found in the ureters in rare instances. The pathological effects which may be produced are dilatation of the pelvis, hæmorrhage, pyelitis, and destruction of the kidney. The symptoms which might be expected are those due to the presence of the worm, which acts as a foreign body, and to its pathological consequences, namely, pain in the renal region, disturbed micturition and strangury, hæmaturia, or pyuria.

3. Bilharzia Hæmatobia—Distoma Hæmatobium.

In Egypt, Cape of Good Hope, and certain other hot countries, this parasite, which was first discovered by Bilharz, is of considerable importance, itself or its ova giving rise to serious consequences. Probably it generally enters the body by the agency of drinking water, or of salads, to which the animal or its embryo or ova adhere. Dr. John Harley has suggested that it sometimes gains access through the skin, in consequence of a person bathing or wading in water, the animal fixing itself and implanting its ova in some superficial vein. Generally an entrance is obtained into the portal system of veins, whence the animal and its ova are conveyed chiefly into the minute veins of the mucous and sub-mucous tissues of the intestines and urinary apparatus. It is in connexion with the latter that the most important effects are produced, and with these we are now concerned.

The Bilharzia is a small trematode, elongated, soft, and bisexual. Its length is about three or four lines, the male being thicker and shorter than the female, which is filiform. The anterior part of the body is flattened, and bears two suckers; the posterior part is cylindrical. The ova are very minute, oval, pointed at one end, or presenting a pointed tooth or spine at the anterior extremity or on one side. The recent embryo is flask-shaped and ciliated. It is supposed that there are two other distinct forms between this embryo and the adult worm, the intermediate stages being passed in fresh-water molluscs and fish.

The morbid conditions induced by this parasite in connexion with the urinary apparatus are chiefly observed in the bladder, but the ureter is also frequently affected, and less commonly the renal pelvis. The ova block up the minute veins, and also make their way through the mucous membrane into the urinary passages. Elevated patches are found on the surface of the mucous lining, consisting of ova mixed with blood, urinary deposits, and crystals of uric acid. Vascular injection, ecchymoses, exudation, and destructive changes ending in ulceration, are also observed. The ureter may be completely blocked up by a mass of ova and urinary sediments, leading to hydronephrosis. Pyelitis may also be excited, followed by pyonephrosis and total destruction of the kidney. According to Griesinger, collections of ova often form the nucleus of a large calculus in Egypt.

The symptoms induced by the Bilharzia will depend upon its effects as above described, and are often very serious. One important point, however, calling for notice is that the endemic hæmaturia which is prevalent in the Cape of Good Hope, Mauritius, and other hot climates is probably due to this parasite. This was suggested by Griesinger, and has been confirmed by the observations of Dr. John Harley in three cases of endemic hæmaturia from the Cape. Numerous ova were found by him in the urine, to which the hæmaturia could be clearly traced. The detection of these ova or their embryos in the urine is the only positive sign of the existence of the Bilharzia disease.

General Remarks on Diagnosis, Prognosis, and Treatment.

It has appeared to the writer expedient, in order to avoid repetition, and to render this part of the subject more comprehensive, to discuss briefly the diagnosis, prognosis, and treatment of growths in connexion with the kidney as a whole. To the more important points bearing on these matters attention will now, therefore, be directed.

1. DIAGNOSIS.—From the account which has been given of the clinical history of the different growths in the kidney, it will be evident that most of them may exist without giving rise to any symptoms which can possibly lead to their diagnosis, and even cancer may be completely latent during life. In some forms, such as the benign growths and the simple varieties of cystic disease, this is almost invariably the case, and it rarely happens that they can be recognized clinically. In other instances obscure renal symptoms are present, but it is impossible to determine their cause with any degree of certainty.

In order to make a complete and satisfactory diagnosis, when this is practicable, it is necessary to take into account the history and age of the patient; the presence or absence of urinary symptoms, and their precise nature, the characters of the urine being of special importance; the signs afforded by any tumour present; the general symptoms and condition of the patient; the state of other organs; and the duration and progress of the case. The degree of difficulty experienced in arriving at a correct conclusion varies much in different cases, some being easily diagnosed, while others are at the best obscure and doubtful. The first point to be determined is whether the kidney is the seat of disease; and then the nature of the complaint has to be made out.

Cases in which there is some new growth in the kidney may be practically divided into, (1) those in which there is an evident tumour present; and (2) those in which no such tumour exists. The former group includes cancer; a simple cyst which attains large dimensions; cystic degeneration; hydatid disease; and, very rarely, a solid non-malignant growth. Tubercular disease does not form a tumour, but an enlargement may be produced in cases of this complaint from the blocking-up of the ureter, and the accumulation of fluids in the renal pelvis; and the same effect may happen as the result of obstruction of this duct by parasites. The chief general characters belonging to renal tumours by which they are distinguished from other forms of enlargement not connected with the kidney, have already been pointed out (see page 609), and this is the first matter to be attended to in the diagnosis. The extraneous conditions with which a renal enlargement is most likely to be confounded are hepatic tumours; enlargement of the spleen; and ovarian or uterine tumours. In exceptional cases difficulty has also been experienced in distinguishing it from fæcal accumulations in the colon, ascites, masses of enlarged absorbent glands, psoas abscess, and aneurism,—the difficulty in the last case being due to carcinoma of the kidney presenting pulsation and a murmur. Without entering into any lengthy description of the distinctive characters, it may be observed that in separating renal tumours from those of other organs, the position of the colon in front of the enlargement is always of great importance. Also they cannot be traced usually within the margin of the thorax on either side, or into the pelvis; and on the right side there is a line of tympanitic percussion-sound between the kidney and the liver, due to the presence of a coil of intestine. In the case of ovarian and uterine tumours, a tympanitic sound is elicited in the lumbar region; whereas if there is a renal tumour, this region is absolutely dull. Vaginal and rectal examination will afford material help in diagnosing these conditions. The chief difficulties arise when a kidney becomes so large as to extend beyond its ordinary limits, perhaps even filling the abdominal cavity; when both organs are enlarged; when a movable kidney becomes the seat of disease; or when a growth or cyst springs from some particular part of the organ, and projects into the regions of other organs. It must also be borne in mind that a tumour of the kidney may exist with other morbid conditions, such as ascites or enlargement of other abdominal organs, and then the diagnosis may be very difficult. Under any circumstances caution is necessary, for renal tumour has always been justly regarded as by no means easy of diagnosis, and serious mistakes have been made by the most competent observers. In addition to other modes of clearing up any difficulty, if the enlargement should become very great, an intelligent patient might be able to indicate where it started, and from what direction it grew, and might thus afford material assistance towards forming a correct opinion.

Renal tumours have in the next place to be distinguished from each other, and from other forms of enlargement connected with the kidney, namely, hydronephrosis, pyonephrosis, abscess in the kidney, and perinephritis. Of course every case must be considered in all its details, before attempting to form an opinion; but, so far as the enlargement is concerned, the chief points to be noticed are whether it is unilateral or bilateral; its extent, dimensions, and rapidity of growth; its superficial characters, and the tactile sensations elicited on palpation and percussion; and whether it is painful and tender or not. The diagnostic characters of hydronephrosis, and of the different forms of purulent accumulation in or around the kidney, have already been discussed in their respective articles. With regard to tumours connected with the organ, an enlargement due to a simple cyst is extremely rare; it is unilateral, painless, and presents a sensation of more or less fluctuation. It would be very difficult to distinguish this condition from hydronephrosis. Congenital cystic disease is observed at birth, and presents the characters already described. Cystic degeneration in adults is very rare; both kidneys are usually affected; the sensation afforded on palpation would probably vary in different parts of the tumour. Cancer is generally readily recognized in infants by the great rapidity of its growth, and the large size it attains; it is also distinguished from congenital cystic degeneration in that it begins after birth. Primary cancer of the kidney is almost always unilateral. The growth is often rapid, and the tumour reaches a large size; it may be nodulated and irregular, and the sensations on palpation are generally those of more or less firmness, while they frequently vary over different parts of the tumour. This is also often painful and tender. The association of such a tumour with repeated and profuse hæmaturia may be regarded as highly characteristic of renal cancer, but it must be remembered that hæmaturia is absent in many instances, while there is a case on record in which an enlarged spleen, associated with leucocythæmia, was accompanied with this symptom. The diagnosis of cancer is likely to be materially assisted by the general symptoms; and perhaps by the discovery of secondary formations in other organs. Non-malignant growths so rarely give rise to a renal tumour, that they might practically be put out of consideration. Such a tumour would be very slow in its progress, never attaining any very large size, unilateral, firm on palpation, and probably painless. Hydatid-tumour is slow in its progress, unilateral, rounded, and fluctuating, and might possibly present hydatid-fremitus, which is highly characteristic. The diagnosis of this disease, however, usually depends on the rupture of the cyst into the renal pelvis and the passage of its contents out of the urinary apparatus. It must be borne in mind that in any case of supposed renal tumour, the aspirateur or the exploratory trochar may prove of the greatest value in its diagnosis, both in determining whether the enlargement really belongs to the kidney, and also in making out its nature.

With regard to the second class of cases, namely, those in which there is no tumour, many of them, including the non-malignant growths and syphilitic gumma cannot be recognized. The most important affection belonging to this group is tubercular disease. Its diagnosis is founded on the presence of pyelitis, not due to any other cause, such as calculus; the special characters of the urine; the general symptoms; and the evidence of implication of other organs, especially the lungs and intestines. If pyonephrosis should arise, the previous history of the case will indicate its nature and cause. Cancerous pyelitis occasionally occurs without tumour, but here, if the urine is altered, it generally contains much blood and but little pus. Hydatid-cysts often rupture before they attain a size sufficient to form an evident tumour, and their existence is then recognized by the escape of their contents. The diagnosis of the other parasitic affections need not be specially alluded to. It must be borne in mind that calculus or other renal disease may be associated with various growths in this organ, and may give rise to their special symptoms. Some of the conditions now under consideration may also burst inwardly in certain directions, and this event would be indicated by corresponding symptoms, according to the direction in which the rupture took place.

2. PROGNOSIS.—This part of the subject may be very briefly discussed. Any actual tumour connected with the kidney may be regarded as involving a serious prognosis, but this differs materially according to the nature of the disease. Any large cyst is liable to rupture; or it may lead to a fatal result by interfering with neighbouring structures, or by suppurating after operation. Cystic degeneration terminates fatally; but in adults the disease lasts a long time, death ultimately occurring from failure of the renal functions. Cancer is necessarily a fatal

disease, and its course is very rapid in children, but much less so in adults. Occasionally the disease seems to come to a stand-still for a time. Hæmorrhage, even when repeated, does not seem to hasten death. Non-malignant growths in the kidney are rarely of much consequence. Tubercular disease is extremely serious, and almost always leads to a fatal termination sooner or later, generally within a short period. Hydatid disease ends very favourably in a large proportion of cases, provided the cyst ruptures into the renal pelvis before it attains any large size. Even after a tumour forms, if the cyst opens into this part, recovery not uncommonly takes place. There is a danger, however, lest it should rupture internally, though even then the issue may be satisfactory, provided the cyst communicates with the intestine; when it has opened into the chest, the result has always been fatal. Suppuration of the tumour is dangerous, but recovery may take place afterwards. If the cyst does not open, but goes on increasing in size, the condition becomes very serious, especially as any operation for the purpose of evacuating it is attended with great danger. The other parasitic diseases are of a very grave nature.

3. Treatment.—But little can be done in the treatment of growths in connexion with the kidney, and it may be confidently affirmed that no medicine can have any direct influence upon them. It has been supposed that hydatid disease could be influenced by turpentine and numerous other drugs, and the parasite killed, but there is no reliable evidence that such is the case. The chief indications to be borne in mind are as follows:—

a. Symptoms connected with the urinary system often require attention. Pain must be relieved by opiates and other anodynes, with warm fomentations or baths. It might be desirable to take away some blood locally. It may be necessary to give remedies to check hæmorrhage, especially in cases of cancer, but in this disease it is not desirable to stop the bleeding if it is only moderate in amount, as the loss of blood may afford relief. Tannic or gallic acid, or acetate of lead, with opium, may be given internally, and ice applied locally. Pyrexia, resulting from pyelitis, needs attention, especially in tubercular disease; and to check the formation of pus full doses of tincture of iron and other astringents may be administered. Symptoms of nephritic colic, due to the passage of blood-clots or hydatid-vesicles, must be relieved by the same means as when they are due to calculus (see page 585.) Mild diuretics have been used to aid the escape of hydatids. When these clots or vesicles reach the bladder, they may be broken down by injections or in other ways, and their escape along the urethra may also be aided. The catheter is sometimes of use, and females have been able to drag out hydatids from the urethra.

b. Operative interference may be called for in some cases. Thus it might be desirable to evacuate the contents of a cyst, and especially of a hydatid-cyst, if this should become dangerously large, and show no signs of opening into the renal pelvis. This evacuation could probably be best effected by means of the aspirateur. Electro-puncture has also been practised with the view of curing hydatid disease, but without success. Irritant injections have likewise been employed. Extirpation of the kidney might possibly be suggested in some cases of cancer or cystic degeneration.

c. Should suppuration take place in connexion with any of these diseases, the treatment must be similar to that indicated when this process arises from other causes. If there is any danger of this event, the patient should be kept entirely at rest, and blood removed locally, or cold applied assiduously.

d. The general condition must be attended to, and any symptoms connected therewith treated as they arise. Good hygienic arrangements and nutritious food are of much importance in cancer and tubercular disease; in the latter tonics, iron, cod-liver oil and such remedies, are also of essential service. Any symptoms apart from the kidney, which result from the renal affection, must also be treated should they occur; and complications must be looked to, including those resulting from the rupture of any accumulation in connexion with the kidney into internal parts.

ANOMALIES OF POSITION, FORM, AND NUMBER OF THE KIDNEYS.

By William Roberts, M.D., F.R.S.

Like various other organs, the kidneys are liable to certain deviations from their natural situation, form, and number. Most of these deviations are congenital, others are acquired at varying periods of life, through accident or disease. They are sometimes capable of being recognized during life, and frequently simulate widely different pathological conditions; many cases escape detection during life so long as the renal functions are satisfactorily performed, and these anomalies may produce little or no inconvenience.

I.—Anomalies of Position.

The kidneys may occupy an abnormal situation and remain permanently *fixed* in that situation, or the misplaced organs may possess a certain degree of *mobility*.

A.—Fixed Malpositions of the Kidneys.

The kidney may be displaced downwards, upwards, or laterally, by the pressure of a tumour growing in its vicinity, or by enlargement of the liver, spleen, pancreas, or supra-renal capsule. In these cases the malposition is *acquired.*

In *congenital malpositions,* the kidney, instead of lying deep in the lumbar region close to the vertebral column, may be fixed in front of the vertebræ, or on the brim of the pelvis, or within that cavity. The most frequent congenital malposition is that where the kidney is found lying obliquely on the sacro-iliac synchondrosis. The kidney has also been found situated beside the uterus, or transversely between the rectum and bladder, or across the prominence of the sacrum.

In those cases where the misplaced organ lies within or upon the brim of the pelvis, it may be felt either through the abdominal wall or through the vagina, and it is of importance to remember the possibility of its being so displaced, lest it should be mistaken for a tumour of a more serious nature. When it is fixed within the pelvis in females, it is liable to embarrass and complicate parturition.

A kidney congenitally misplaced usually deviates more or less from its natural configuration and is associated with malposition of some portion of the large intestine and peritoneum. The renal artery and ureter also necessarily deviate more or less from their normal distribution. Ruysch figures a case in which the kidney lay crosswise with its hilus turned upwards, and the ureter descending behind it. The corresponding supra-renal capsule never accompanies the kidney in its misplacement in congenital cases, but is invariably found to occupy its usual situation in the lumbar region.

In twenty-one cases of congenital malposition which I have been able to collect and compare,[1] the abnormality was in every instance confined to one kidney. The left kidney was much more commonly affected than the right, fifteen out of the twenty-one cases occurring in the left kidney, and six in the right.

It rarely happens that malpositions of this class show any evidence of their existence during life. A curious specimen has been described and figured by Mr. Canton.[2] It was taken from the body of a man who died from bronchitis at the age of twenty-seven, in whom no renal symptoms were manifested during life. The right kidney was in every respect normal, but the left was situated in the angle formed by the bifurcation of the aorta, and instead of presenting the ordinary kidney-shape, it was irregularly oval and lobulated on some parts of its surface. The pelvis of the organ was directed almost immediately forwards, and there was dilatation of the upper portion of the ureter owing to the impaction in it of an oxalate of lime calculus weighing two and a half drachms. The left renal arteries were two in number, and took their origin from the anterior portion of the aorta a short distance above its bifurcation. The sigmoid flexure of the colon was placed on the right side of the kidney.

Mr. Durham[3] relates a case in which the misplaced organ formed "a tumour of doubtful character," deeply seated in the hypogastric region, somewhat on the left of the middle line. The patient ultimately died of pulmonary disease, and at the autopsy this supposed tumour was found to be nothing more than the left kidney which was situated over the sacro-iliac synchondrosis, extending over the sacral promontory and into the true pelvis. The kidney presented no distinct hilus and had not the characteristic kidney-shape. The ureter was formed by the junction of four branches, two from the upper and posterior part, and two principal ones from the lower and anterior part. The right kidney and both supra-renal capsules were in the normal position—both kidneys were healthy. The colon formed no sigmoid flexure in the left iliac fossa, but passed across the middle line; and the commencement of the rectum was on the right side of the sacrum. In a case under the observation of Hohl, cited by Rayer,[1] the left kidney was deeply situated on the inside of the psoas muscle, and during two previous labours had retarded the passage of the fœtal head; on both occasions, however, delivery was safely completed.

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[1] For two additional cases of fixed malposition of the kidney, see Hausmann, Monatsschr. f. Geburtsk. xxxiii. p. 401, and Gosselin, L'Union Méd. 1869, p. 115.

[2] Path. Soc. Trans. xiii. p. 147.

[3] Guy's Hosp. Reports, 1860, p. 407.

The *diagnosis* of a misplaced kidney, forming a pelvic or abdominal tumour, is based on the moderate size and smooth elastic feel of the tumour, together with the existence of a slight hollowing or want of fulness of the corresponding lumbar region, denoting the absence of the kidney from its normal situation. When the tumour is reniform, its shape materially aids the diagnosis as a matter of course; in a large majority of such malpositions, however, the characteristic kidney-shape is not preserved.

B.—Movable Kidneys.

SYNONYMS:—Floating Kidney; les Reins Mobiles, les Reins Flottants (Fr.); Bewegliche Niere (Germ.); Renes Mobiles (Lat.); Rene Mobile (Ital.).

HISTORY.—Although vague allusions to mobility of the kidneys are met with in the writings of the older writers, especially in those of Mesué and Riolan,[2] the merit of having first pointed out the practical bearings of this condition, and the signs and symptoms by which it may be recognized during life, is due to Rayer.[3] In this country the subject has been ably illustrated by Dr. Hare;[4] and Mr. Durham has, in his paper previously alluded to, brought together and collated ten cases, being the total number which had been verified by post-mortem examination up to the time he wrote (1860). In Germany, Oppolzer[5] and Henoch[6] have contributed a number of cases, and Fritz[7] has analysed all the published cases up to 1859. Still more recently the comprehensive essays of Becquet[8] and Rollet[9] have yet farther elucidated the subject. Finally, the present writer[10] has collated seventy cases, partly from the sources indicated above, and partly contributed by himself. On an analysis of these seventy cases the following account is based:—

PHYSICAL SIGNS AND SYMPTOMS.—In the normal condition the kidneys are retained in their position by a thick investment of adipose tissue and by a reflection of the peritoneum, which passes over their anterior surface. It can easily be understood how under certain circumstances one or both kidneys are liable to break away from these somewhat lax attachments and float loose among the abdominal viscera, being retained only by their blood-vessels and excretory ducts. The degree of mobility and of change of position which the kidney acquires in these cases varies considerably. When the patient stands upright, the organ in the majority of cases descends below the margin of the ribs and forms an oblong tumour of the shape and feel of the kidney, occupying a diagonal position extending from below upwards and outwards, midway between the umbilicus and the costal border. It can be pushed in various directions—upwards, downwards, or laterally—over a space of several square inches. In a case observed by myself inflammatory adhesions had taken place to the surrounding structures, and the organ had become permanently fixed in its new position. The displaced organ is usually painless, but handling or compressing it, which can be easily practised in thin persons with flaccid abdominal walls, gives rise to peculiar sensations of nausea or sinking.

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[1] Maladies des Reins, tome iii. p. 774.

[2] Manuel Anatomique et Pathologique, Jean Riolan, 1682.

[3] Op. cit.

[4] Med. Times and Gaz. 1858, i. p. 7, &c., and 1860, i. p. 30.

[5] Ibid. 1857, i. 575; and Clin. Européenne, 1859, No. 2.

[6] Klinik d. Unterleibs-Krankheiten. Berl. 1858, Bd. iii. p. 367.

[7] Archives Générales, Août et Sept. 1859.

[8] Ibid. Jan. 1865.

[9] Zur Pathologie u. Therapie d. bewegl. Niere. Erlang. 1866.

[10] A Practical Treatise on Urinary and Renal Diseases, 3rd edition; London, 1876, p. 607.

When the patient lies in the horizontal posture the kidney can be replaced in its normal site in the lumbar region, but as a rule, as soon as the pressure is withdrawn it resumes its abnormal situation.

The position of a movable kidney is greatly influenced by the respiratory movements and by the posture of the body; thus deep inspiration causes it to descend, and deep expiration to ascend, and the force of gravity drags it to whatever side the patient's body is inclined. In the slighter cases half or three-quarters only of the length of the organ can be felt through the soft abdominal walls along the borders of the false ribs. Most commonly, however, the displacement is to a much greater extent. In a case mentioned by Johnson the kidney had drifted below the umbilicus, and in another related by Day, it lay in the iliac fossa and could be moved over an area of three or four inches; in a case under my own observation the organ could be felt lying on the edge of the pelvis in the vicinity of the cæcum.

When the patient is recumbent the displaced kidney occupies a higher position than after long standing or walking. Percussion over a movable kidney yields not a dull sound, but a muffled tympanitic note. On examining the loins, a slight hollowing or flattening of the renal region is observed on the side of the displacement, and the percussion sound is tympanitic, indicating that the intestine occupies the normal site of the kidney: if the organ be reinstated in its original position, the natural prominence in the lumbar region is restored, and the bowel sound disappears.

The subjective symptoms vary considerably. Occasionally the symptoms are so slight and obscure that the attention of the patient is not drawn to the anomaly; and it has on more than one occasion been detected by an accidental examination of the abdomen during the course of some other complaint. As a rule, however, considerable suffering and inconvenience result from the displacement. The most constant symptom is a dragging pain in the affected side aggravated by walking or standing; this is of a neuralgic character and radiates from the neighbourhood of the displaced organ into the loins, round the waist, down to the hypogastric region, and along the thighs.[1] Sometimes the sufferers are conscious of the existence of a movable tumour in the abdomen, and this occasions great mental distress and not unfrequently leads to a condition of confirmed hypochondriasis. In one case the movements of the displaced kidney were mistaken for those of a child in the womb. Sometimes there is disturbance of the gastric functions, more frequently the bowels are affected either with fitful diarrhœa or with constipation; whilst in other cases there are occasional attacks resembling biliary or nephritic colic, accompanied with nausea, vomiting, shivering, faintness, and signs of local peritonitis; during such attacks the kidney becomes swollen and forms an immovable, painful tumour in the abdomen. In these cases the kidney generally resumes its former size and mobility on the subsidence of the paroxysm, which usually takes place after a few days' rest in bed and the use of opiates and warm local applications; but occasionally inflammatory adhesions are formed, and the kidney remains permanently fixed in its new position.

The origin of these attacks is somewhat obscure: they sometimes set in suddenly while the patient is in bed, without any appreciable cause; more frequently, however, they follow some unusual exercise or an indigestible meal, or they occur at the menstrual periods. It is not improbable that, at least in some cases, they are due, as to a kind of strangulation of the kidney, from the pressure of the displaced organ on its own ureter, and a consequent obstruction to the flow of urine, leading to acute engorgement of the organ, with dilatation of the pelvis and pyelitis. This hypothesis seems to derive support from the observation of Rollet,[2] that the urine sometimes becomes bloody and purulent for some time after one of these attacks.

The excretion of urine, which is always quite healthy, generally remains unaffected; occasionally, however, micturition is unnaturally frequent and accompanied with more or less pain.

Epigastric pulsation is a frequent concomitant of mobility of the kidneys, and in three cases this was a prominent symptom. In four cases hydronephrotic distension of the renal pelvis coexisted with mobility of the kidney. Among other complications which have been observed in these cases may be mentioned Bright's Disease, the discharge of uric acid gravel, œdema of the lower limbs from compression of the ascending cava by the displaced kidney, and obstinate constipation from similar compression of the colon.

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[1] In two cases under the observation of Rayer (op. cit.) the pain extended into the right ham, along the posterior part of the thigh and even as far as the ankle. It sometimes seemed to start from the right loin and extend into the labia majora.

[2] Loc. cit. p. 20.

ETIOLOGY.—Mobility of the kidneys is much more common in women than in men, and on the right side than on the left. Thus of the seventy cases collected by the writer, sixty-one occurred in women and only nine in men. Information as to the kidney affected is given in sixty-five cases—in 42 of which the right alone, and in 9 the left alone was movable; while in 14 cases both kidneys were movable. The *age* of the patients ranged between sixteen and sixty-five years—the greatest number being between twenty-five and forty, thus corresponding roughly to the child-bearing period in women.

Judging from my own personal experience, this affection is much more common than is generally supposed; and I am inclined to the belief that many cases of obscure abdominal pain and gastro-enteric disturbance are due to this cause. A large number of cases are undoubtedly overlooked. Rollet states that out of 5,500 patients admitted into Oppolzer's clinique and examined carefully with regard to this anomaly, 22 had movable kidneys—a proportion of one in 250 cases.

In a certain number of cases, no clear determining cause can be discovered; but as a rule the antecedent history of the patient discloses some circumstance or circumstances to which this anomaly can be attributed. The displacement is due in many cases to repeated or protracted labours, and this partly explains the greater frequency of movable kidneys in women than in men: the alternate tension and relaxation of the abdomen, and the convulsive muscular efforts accompanying parturition, must obviously have a tendency to loosen the attachments which hold the kidney in its place and favour its migration under the force of gravity into a lower position in the abdomen.

The disproportionate frequency of movable kidney in the female sex, and especially on the right side, is in part due to tight-lacing. With reference to this point, Cruveilhier[1] observes: "I have often observed in women who wore tight stays, the right kidney to lie sometimes in the right iliac fossa, sometimes in front of the sacro-iliac synchondrosis, sometimes even in front of the vertebral column at the level of the adherent border of the mesentery, in the substance of which it was placed. The kidney thus displaced enjoys a certain mobility. This displacement of the kidney arises when the pressure exercised on the liver by the stays dislodges the right kidney from the kind of niche which it occupies on the under surface of this organ.

"If the left kidney is not so frequently displaced as the right, that is owing to the fact that the left hypochondrium occupied by the spleen and the great end of the stomach bears the pressure of the stays with much more impunity than the right."

It seems probable that in some cases mobility of the kidneys has been determined by rapid emaciation and the removal of the capsule of adipose tissue which naturally invests the kidney; thus Oppolzer invariably found a deficiency of the cushion of fat about the kidney in the cases examined by him, and in a case dissected by Mr. J. Adams[1] "the only peculiarity remarkable was that the kidney appeared bound down more loosely than usual, and the old lady, from having been very fat, had become somewhat thinner, and her integuments appeared very lax throughout."

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[1] Traité d'Anatomie Descriptive, tome iii.

In a considerable number of cases, more especially in men, displacement and mobility of the kidney is produced by a blow on the loin, a sudden fall or jump, violent running, dancing, riding, or some inordinate muscular effort[2] or succussion of the body. These causes generally produce their effect suddenly, or sometimes more slowly, as in one of Henoch's cases, where no symptoms supervened until about half a year after a fall from a horse.

Rayer relates the case of an old woman whose right kidney was displaced and movable, apparently from its being dragged down, or at least left free to descend by its own weight, in consequence of displacement of the peritoneum from a hernia of the cæcum. It is probable that the hydronephrotic condition which was found in four of the recorded cases acted in a similar way in dragging the kidney from its normal site by the pressure of the distended pelvis and consequent increase in the weight of the organ.

Becquet[3] has enunciated a novel theory to explain the liability of women to mobility of the kidneys. He says: "On the breaking forth of the menstrual flux, the kidneys are associated in the congestion of the generative organs and become swelled. This fact, less rare doubtless than is usually supposed, perhaps even physiological, does it not explain the renal pain so often felt at the menstrual periods, especially in women who are subject to dysmenorrhagia? Thus swelled and rendered heavier, the kidney, and especially the right kidney, strains the feeble attachments which retain it, and tends to start out of its place. Soon the congestion subsides, and the organ returns to its original position; a second congestion displaces it further; and a third still more: the kidney, becoming each time heavier from the incompleteness of the resolution, comes to occupy a lower position; and thus gradually and at length, but not without suffering, breaks loose and floats in the abdominal cavity." In a case under the observation of the writer, the displaced and movable kidney seems to become larger two days before the catamenia appear; it becomes sensitive to the touch, and at times somewhat painful even without being touched; and in a case observed by Dr. Ritchie and cited by the writer, an opportunity was afforded of watching the condition of the kidney during two menstrual periods, when its size was seen to be increased by fully one-half, and it was much more sensitive to the touch. These facts, so far as they go, lend an air of feasibility to the theory of Becquet.

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[1] Med. Times and Gaz. 1857, i. p. 651.

[2] In two cases observed by the writer, the displacement was perceived soon after a violent attack of bilious vomiting.

[3] Arch. Générales, 1865, i. p. 21.

Hitherto we have been discussing only those cases where the mobility of the kidney appears to be due to circumstances arising after birth and where there is generally evidence of the present or past existence of some obvious cause of displacement and mobility. The majority of cases belong to this category, but mobility of the kidneys may be congenital; when so, it is commonly associated with an anomalous arrangement of the peritoneum, or with an abnormal origin and distribution of the renal vessels.

In two cases observed by Dr. Priestley[1] and M. Girard[2] respectively, the peritoneum was found reflected over the posterior as well as the anterior surface of the kidney, so as to enclose it within its folds, thus forming a distinct meso-nephron and allowing it very considerable motion in the abdomen. In the body of a woman, examined by Mr. Durham, mobility of the left kidney was due to an abnormal arrangement of the peritoneum. The peritoneum instead of passing over the anterior surface of the kidney, only just touched its lower border, and after having formed the descending meso-colon, again touched its outer border. The lesser sac of the peritoneum also passed so far to the left as to cover the posterior surface of the spleen, and so far downwards as to touch, and be reflected from, the upper border of the kidney. There was thus no distinct meso-nephron; but the kidney, instead of being supported and retained in position by a single layer of peritoneum, was left free to move between and beneath three diverging layers.

THE DIAGNOSIS as a rule is easy; and errors have arisen rather from the possibility of such a condition not having occurred to the practitioner, than from any inherent difficulty in detecting it.

The diagnosis chiefly depends on the presence of a hard, smooth, slippery, movable tumour having the size and shape of the kidney, or approaching thereto, on one side of the abdomen, generally in the hypochondriac region; it can at will be pushed with the thumb and fingers into the lumbar space, and out of it again. When the corresponding loin is examined, some degree of flattening or hollowing can usually be detected in the normal site of the kidney, and on percussion over this hollow, a tympanitic sound is elicited. The diagnosis may however be embarrassing in obese people and where the mobility and malposition are slight. It is necessary to remember that the displaced organ may contract adhesions in its new position; these will of course restrict the area over which it can be moved.

TREATMENT.—The most obvious indication is to replace the kidney in its normal site, and retain it there; if this cannot be effected on account of adhesions, endeavours must be made to maintain it steadily in its new position, so that it shall not drag asunder its new attachments[1] by its own weight. These objects are attained by the application of a belt or bandage round the abdomen, after the kidney has been replaced in its proper position. This with careful regulation of the bowels (as any fæcal accumulation aggravates the inconveniences of this affection) is frequently all the treatment necessary. Sometimes, however, more steady pressure and support can be afforded by means of a pad fitted over the situation where the kidney usually protrudes, in addition to the belt or bandage. These mechanical appliances cannot in every case be borne, but usually relief, partial or complete, is afforded by them.

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[1] Med. Times and Gaz. 1857, i. p. 263.

[2] Journal Hebdom. No. 53, p. 445, cited by Rayer, op. cit.

If anæmia or any other constitutional vice be present, it must be remedied by appropriate treatment. Restoration of the tone of the abdominal muscles which are generally relaxed and flaccid, is probably the most effective means of reducing to a minimum the discomforts associated with movable kidneys. For this purpose, ferruginous and other tonics and shower baths, with the avoidance of tight lacing and all violent modes of exercise such as equitation, dancing, and the like, are the best means to be used.

When symptoms of so-called strangulation of the kidney supervene —violent pains, sickness, frequent micturition, enlargement and excessive tenderness of the tumour—complete rest in the recumbent posture, hot poultices or even leeches applied over the seat of pain, and the hypodermic administration of morphia, are the measures most likely to afford relief.

II.—ANOMALIES OF FORM.

Deviations from the normal shape of the kidneys may be *congenital* or *acquired* at different periods of life, as a result of the pressure of tumours or morbid enlargements of neighbouring viscera. Some of these abnormalities have been already adverted to incidentally, in the article on fixed malpositions of the kidney. The lobulated character of the gland, which is the normal condition during fœtal life, sometimes persists, to a greater or less extent, during the remainder of life. Depressions on the surface of the kidney are not uncommon, dividing the organ into two or three indistinct irregular portions. Sometimes although both kidneys are perfectly healthy, one is two or three times larger than the other; this arises probably from deficient development of one renal artery.

Curious anomalies in the structure and development of the pelvis of the kidney and the ureter are occasionally encountered; thus in a case recorded by Sir Henry Thompson,[2] the kidney was provided with two pelves which united so as to form a single ureter about an inch below their necks. Mr. Wood[1] relates a case in which the left kidney had two ureters which remained distinct until within an inch of the bladder. The right kidney of the same patient, in addition to a ureter which entered the bladder at the usual place, was provided with an aberrant ureter which was sacculated and about the thickness of a goose-quill; it was connected with a dilatation (partial hydronephrosis) at the upper extremity of the kidney, and opened into the bladder close to the vesical orifice of the urethra. Dr. C. Kelly[2] records a case of solitary right kidney with two ureters, the upper of which descended in the usual manner, while the lower one passed behind it, and following the course of the left common iliac artery, entered the bladder in the usual position of the left ureter. There were three arteries, the upper of which occupied the usual position of the right renal artery, and three veins; no corresponding vessels existed on the left side.

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[1] Rollet (op. cit.) states that in cases where the diagnosis is undoubted and the adhesions recent, the kidney may be forcibly pushed into its normal position and the affection be thus at once and permanently cured.

[2] Path. Soc. Trans. vol. vi. p. 267.

Horse-shoe Kidney.—This abnormality consists in the coalescence of the two kidneys into one by means of an intermediate transverse portion, which connects their lower ends across the front of the vertebral column, so as to form a crescent or horse-shoe with the concavity directed upwards. The two halves of a horse-shoe kidney are usually complete and perfect in themselves, having each a distinct pelvis and ureter. The isthmus usually consists of proper secreting structure, but sometimes it is composed merely of condensed fibrous tissue. Most frequently the ureters descend in front of the transverse portion, but more rarely they pass behind it; in a drawing in the possession of my colleague, Dr. Renaud, the two ureters are seen crossing each other on their way to the bladder. In thin persons a horse-shoe kidney may be felt through the abdominal walls—and may thus be mistaken for a morbid growth.

No interference with the renal functions results from this abnormality; but it is well to remember the possibility of its occurrence, for if dilatation of the pelvis of such a kidney took place, the central position of the tumour near the spine would be very liable to direct the attention of the observer away from the idea of hydro- or pyonephrosis, if the possible existence of this "horse-shoe" condition was overlooked.

III.—ANOMALIES OF NUMBER.

Supernumerary Kidneys.—A number of instances are cited by Rayer in which there existed one or two supernumerary kidneys, each with its separate excretory duct.

Solitary Kidney.—It has been repeatedly observed that only a single kidney has been found in the bodies of persons who during their lives had suffered from no derangement of the urinary function. In such cases, the existing kidney is always hypertrophied, and no interference with the secretion of urine takes place so long as it remains healthy; but should it become the seat of inflammation, or should its excretory duct become obstructed from the pressure of a tumour, or the impaction of a calculus, partial or total suppression of urine takes place accompanied by very alarming symptoms, and ending in fatal uræmia.

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[1] Path. Soc. Trans. vol. vii. p. 261.

[2] Ibid. vol. xix. p. 274.

Mosler and Rayer have recorded a number of cases of solitary kidney, and the writer has collected numerous others, amounting in all to twenty-nine cases, of which twenty-two occurred in males, six in females, and in the report of one the sex is not mentioned. One was a male infant seven days old, another a boy of seven years; two of the cases were fifteen years old, four between twenty and thirty, three between thirty and forty, four between forty and fifty, two were sixty and one sixty-five at the time of death—the remainder occurred in adults whose age is not recorded.

The left kidney was absent in sixteen cases, and the right in twelve, while in one case the side is not mentioned.

The defect was congenital in nineteen cases, while in three it had been acquired during life through the previous destruction of one of the organs; in seven cases it could not be determined whether it was congenital or acquired.

The renal vessels and ureter of the defective side were always, and the corresponding supra-renal capsule generally, wanting when the defect was congenital. In Dr. Hillier's case, the aorta gave off three arteries to the existing (right) kidney, which was provided with two ureters; the left supra-renal body was in its normal situation.

The cause of death was specified in twenty-four of the recorded cases. The impaction of a calculus was the most frequent cause, as it led to a fatal issue in ten of these cases, while inflammation of the kidney produced death in three, and phthisis in two cases. Death resulted in one case from each of the following causes: pressure of a cancerous tumour on the ureter, impeded flow of urine from congenital phymosis, valvular disease of the heart, peritonitis, meningitis, double pneumo-thorax, typhus fever, "accidental injury," and rupture of the kidney. In an epileptic girl under the care of my colleague, Dr. Leech, solitary kidney was associated with double uterus and vagina.

Absence of both Kidneys.—Rayer has collated a number of examples of this anomaly in still-born children, there being entire absence of both kidneys together with the ureters and bladder. This abnormality seems to be not uncommon in anencephalous monsters.

DISEASES OF THE URETER.

By Frederick T. Roberts, M.D., B.Sc., F.R.C.P.

Most of the morbid conditions to which the ureter is liable have already been incidentally alluded to in some of the previous articles on Diseases of the Kidney, and the consequences have been discussed; it will now, therefore, be only requisite in the first place to give a concise summary of these morbid conditions, and to indicate their nature; secondly, to point out the signs by which they are recognized clinically, and the effects which they produce; and thirdly, to offer a few observations on their treatment.

A.—Summary of Morbid Conditions.

1. Congenital Malformations.—The ureter is occasionally found to be completely impervious, being converted into a solid cord; or its lower opening is closed, or is surrounded by a dense fibrous ring. Again, it may open into the renal pelvis in an unusual manner, entering at an acute angle, and presenting a valve-like arrangement, which prevents the escape of the urine. One of the most common malformations is the presence of two ureters in connexion with one kidney, which may be associated with a divided pelvis, and the two ducts may open separately into the bladder, or join before they reach this viscus.

2. Injuries.—The ureter has in rare instances been ruptured across as the result of a crushing accident. It has also occasionally been torn in connexion with a gun-shot wound. Lesser degrees of injury have been supposed to set up inflammation and its consequences in some cases. Under this head may also be mentioned the fact that the ureter sometimes ruptures as a consequence of injury from within, due especially to calculus, which either directly destroys the wall of the tube, or first originates ulceration, and this subsequently leads to perforation. This tube has also been cut across during the performance of operations, such as ovariotomy.

3. INFLAMMATION.—This condition of the ureter is often associated with inflammation affecting the bladder or renal pelvis, or it may be independent, originating from irritation of the inner surface of the tube by foreign bodies, especially calculi, or possibly from external injury. The lining membrane is reddened and swollen, and it may present flakes of lymph, or yield a muco-purulent fluid. In some cases more or less ulceration ensues. In others the walls of the ureter are infiltrated and thickened, and they may become very firm as the result of a chronic inflammatory process.

4. MORBID GROWTHS.—The ureter is liable to be affected with cancer, when this disease implicates other parts of the urinary apparatus. It seems to have been chiefly observed as the result of extension from the bladder. Tuberculous disease also generally involves the ureter along with the kidney and bladder. A deposit of so-called scrofulous material forms; the mucous lining becomes destroyed or ulcerated more or less extensively, with proportionate discharge; the walls are frequently much thickened and indurated; and the canal is considerably narrowed. In cases of the affection due to the Bilharzia Hæmatobia, the ova of this parasite are found in the ureter, either alone or mixed with urinary gravel and uric acid crystals. They constitute irregular and distinct patches, slightly elevated, of a greyish-yellow colour, soft, but firmly adherent.

5. OBSTRUCTION.—The obstruction of the canal of the ureter is highly important, and it may arise in the following ways:—(*a*) As the result of morbid changes in the walls, in connexion with some of the conditions already alluded to. Thus the mucous lining may be swollen, or a deposit formed upon it; the entire walls may become thickened and infiltrated; or cicatrization may take place after ulceration. In either of these ways the channel of the ureter becomes more or less encroached upon, and it may even be completely obliterated in some part of its course. This obliteration may also be congenital. (*b*) From internal obstruction by foreign bodies or morbid products. Of these the most important is a calculus, which only temporarily blocks up the tube, or becomes firmly impacted in it; but obstruction may also arise from fragments of cancerous or tuberculous matter, hydatid-vesicles, blood-clots, or inspissated pus. (*c*) From external compression. The ureter is liable to be pressed upon from the outside by tumours connected with the kidney itself, by abnormal renal vessels, or by neighbouring growths, such as an ovarian cyst or an uterine tumour. Under these circumstances its canal may also be completely closed. (*d*) From twisting of the duct upon its own axis. This is a rare cause of obstruction of the ureter.

6. DILATATION.—As a consequence of any of the causes of obstruction just mentioned, the ureter is liable to become more or less dilated above the seat of such obstruction, and at the same time frequently

becomes lengthened and tortuous. Moreover, if several concretions or other bodies pass down the ureter in succession or at intervals, the tube tends to remain permanently dilated, so that the passage of these bodies becomes less difficult in course of time. The ureter may also be distended along with the other portions of the urinary apparatus, in consequence of some obstruction affecting the canal of the urethra. The extent of the tube which is dilated depends upon the seat of obstruction, when the enlargement is due to this cause. The dilatation is usually not uniform, but unequal, and a sacculated appearance is consequently produced. The degree of distension also varies; in several instances the ureter has become so enlarged as to resemble the small intestine. Generally its walls are thickened, and the muscular tissue may be hypertrophied; on the other hand, atrophy is occasionally observed, and the tube has even been known to have undergone such attenuation that it has ultimately given way. Below the seat of any obstruction, the ureter generally becomes narrowed and wasted.

7. Hypertrophy and Atrophy.—These conditions need merely be mentioned, as they have been sufficiently noticed above. Thickening of the walls of the ureter from inflammatory infiltration must not be confounded with true hypertrophy.

B.—Symptoms and Effects.

The principal clinical phenomena which may be observed in association with the morbid conditions of the ureter which have been indicated in the preceding remarks, are attributable to their effects, and are not immediately connected with this tube. In some instances there will be no symptoms at all, but as a rule a knowledge of the condition present may be derived from a due consideration of the signs revealed by a careful clinical examination. The phenomena may be summarized as follows:—

1. The ureter is liable to become the seat of painful sensations. Probably inflammation or ulceration of this tube may be accompanied with more or less pain or uneasiness in its course. It is, however, in connexion with the passage of calculi or other materials from the pelvis of the kidney to the bladder, that the severe suffering is associated which constitutes renal or nephritic colic, the characters of which have already been described under Calculous Disease. The rupture of the ureter may be attended with a sudden pain, accompanied with the feeling of something hot being poured out. Even when there is no spontaneous pain, tenderness may be felt in some cases along the course of this tube.

2. One of the most serious effects of many of the morbid conditions of the ureter is that obstruction is produced, either temporarily or permanently, and consequently urine or morbid fluids which form in connexion with the kidney cannot escape, but accumulate above the seat of obstruction. Hence follows in different cases hydronephrosis or

pyonephrosis, with their several consequences, and the clinical signs of these conditions become the data upon which a knowledge is founded of what is taking place in connexion with the ureter. Should the cause of the obstruction be got rid of, the fluid collection will escape suddenly, and, as has already been pointed out several times, the tube may be alternately blocked up and free, with corresponding changes in the hydronephrosis or pyonephrosis. Concretions of phosphate and carbonate of lime are also liable to form in the renal pelvis and calices, if the ureter is rendered impervious in any way.

3. It may be mentioned that in some affections the ureter itself forms morbid products, such as pus, and these become mixed with the urine; it is quite impossible, however, to distinguish this admixture as coming specially from the ureter, as there are always similar products derived from the renal pelvis, and they cannot be separated. A similar observation applies to the escape of blood from the ureter.

4. The rupture or perforation of the ureter is attended with grave consequences. The urine escapes, and sets up violent inflammation in the parts around, leading to the formation of abscesses or to peritonitis, with the accompanying symptoms, both local and general. The termination is always fatal.

5. Dilatation of the ureter may in some cases of renal calculus be recognized by the fact, that when a number of concretions are passed in succession or at intervals, the attendant suffering and other symptoms become markedly diminished, on account of their more easy passage along the dilated tube. This may, therefore, be a favourable sign in such cases. Dilatation from other causes cannot be definitely detected.

6. Physical examination by manipulation along the course of the ureter may enable certain conditions associated with this tube to be discovered. Thus a calculus lodged in it may occasionally be felt, or its passage along the duct may be traced. Possibly, also, if the ureter is much dilated and hypertrophied, it might be recognized by careful manipulation through the abdominal walls.

7. General or sympathetic symptoms are not uncommonly associated with diseases affecting the ureter. As illustrations may be mentioned the symptoms accompanying the passage of a calculus; more or less collapse, followed by pyrexia, in cases of rupture of the ureter; and the wasting, hectic, and night-sweats observed in connexion with tuberculous disease.

C.—TREATMENT.

It is not often that any direct treatment can be applied to the ureter, and the objects to be borne in mind may be stated in a few words. The first and most important point is to assist the passage of any foreign body along the tube, if lodged there; to relieve the accompanying symptoms; and to prevent any injurious effects. These indications are carried out by keeping the patient at rest; the administration of opium or other anodynes; the use of warm fomentations or baths; the local removal of blood in some cases; and by careful manipulation along the course of the ureter. (See Calculous Disease.) If anything is known to be pressing upon the ureter, this also must be got rid of, if possible. Often, however, nothing can be directly done to remove obstruction affecting this tube. Under such circumstances the treatment must be directed to the effects of this condition which are associated with the kidney, such as hydronephrosis or pyonephrosis. If there are signs of inflammation of the ureter, hot fomentations should be freely used, or in some cases it might be advisable to apply a few leeches along its course. Nothing can be done in cases of rupture of the ureter, except to keep the patient absolutely at rest, and to treat the consequences of the lesion. General treatment is indicated in certain forms of disease, similar to that described as applicable to the same affections when they involve the kidney, of which they form a part.

AFFECTIONS OF THE BLADDER.

BY SIR HENRY THOMPSON, M.B., F.R.C.S.

dder is subject to numerous maladies; some of them are of ı occurrence. Not a few are associated with conditions either ›rgan itself, or of some adjacent part, which require mechanical nt, and therefore fall naturally under the care of the surgeon. e tumours of the bladder, calculus, hypertrophy of the prostate, e of the urethra, etc., all of which may demand the use of ıents. For other maladies, on the contrary, such treatment is y not necessary, but would be prejudicial if employed. It is l therefore that the physician should understand clearly when t solely by medicine and its adjuncts, and when to invoke geon's aid. Hence, to consider, as far as it is possible to n somewhat narrow limits, the diagnosis of this group of s, as well as to advise the medical treatment, will be the aim present sketch.

ollowing table comprises the principal affections which it will ssary to allude to in the following pages:—

mmation of the bladder—acute, subacute, or chronic catarrhal.
in the bladder.
ıtinence of urine.
ıtion of urine.
y and over-distention of the bladder.
ysis of the bladder.
ıic changes in the bladder—hypertrophy, sacculation, tumours
wths.

AMMATION OF THE BLADDER—ACUTE.—This affection is manin two distinct forms, differing considerably in gravity and y; yet both are essentially acute, since in each the invasion en, the progress is rapid, and fever is present. The more severe mostly due to traumatic causes, such as surgical operations, uries; to calculus; to the later stages of prolonged retention e from obstructions in the urethra; and to chemical agents, as ides, &c. It may also occur by continuity from inflammation prostate, or of the kidneys. The symptoms are excessive frequency and pain in passing water, pain about the loins, pelvis, and perineum, and in the penis; tenderness to pressure above the pubes and in the rectum. The urine is loaded with inflammatory products, mucus, pus, and blood, and often shreds of lymph in some quantity; the blood increasing rapidly as the disease advances. Considerable fever is present, often marked by repeated attacks of rigors, which are followed by heat and then by profuse sweats.

The less severe form often arises from gonorrhœal inflammation spreading backwards, from exposure to cold, from chemical irritants; sometimes in peculiar idiosyncrasies, perhaps occasionally from gout, and even from indigestion. The symptoms are as follows: first, frequent micturition, inability to retain the urine for an instant when the want to pass it is experienced. Urine previously clear becomes cloudy from admixture of simple mucus, not to be confounded with opacity produced by the pressure of lithates or of amorphous phosphates, the former disappearing by heat, the latter by the addition of nitric acid; often there is a moderate degree of fever; the patient has usually accelerated pulse, loss of appetite, and thirst. Some pain is generally felt above the pubes, in the perineum and sometimes in the urethra, and micturition is also in some cases painful.

Treatment of the two forms.—First, it is necessary to ascertain if retention of urine is present, since it may be a cause or a serious complication of the malady. The presence of retention may be suspected if there is difficulty in expelling the urine, as well as great frequency in the attempts to pass it, and if the secretion is alkaline; but the condition can only be determined by examining the region. There may be a swelling in the situation of distended bladder above the pubes, dull to percussion, and distinguishable by a finger introduced into the rectum; whether or no, this important question can only be solved by passing a catheter with the utmost care and gentleness. Supposing, however, a case in which the cystitis is not due to surgical operation and is not one requiring mechanical relief, the following is an outline of the treatment. Hot fomentations or poultices to the suprapubic and perineal regions; hot hip-baths or bidets. Gentle action of the bowels is to be insured by mild aperients; diluent drinks should be taken freely, and frequent small doses of alkali, full doses of henbane, or small doses of morphia or chlorodyne. Suppositories of morphia or of opium are very useful when pain and irritability of the bladder are extreme. When the attack is subsiding, or if it is slight throughout, the patient may take three or four ounces of the infusion of buchu three times a day, or a pint of strong decoction of triticum repens daily.[1] No alcoholic stimulants should be permitted, but the food should be nutritious, light, and digestible.

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[1] The best formula is the following: boil four ounces of triticum repens in a quart of water, gently reducing it to a pint: strain and cool: let a third part be taken three times a day.

CHRONIC CYSTITIS.—This affection in a mild form often appears to be associated with the existence, as a primary affection, of morbid changes in the kidneys; and sometimes follows the formation of renal calculus. Its presence is also very commonly the sign of obstruction in the urethra, or of calculus; more frequently still of prostate hypertrophy; rarely, of tumour of the bladder. Many of the symptoms do not speedily yield to medical treatment, the presence or absence of these conditions must be verified by a practised hand. For it is never to be forgotten that in chronic cystitis, the source of the disease is almost always revealed by a physical examination. Its most frequent causes are the following: chronic retention of urine from obstructive disease of the urethra (stricture in early and middle life, hypertrophy of prostate in advancing years); atony of the bladder, occasionally paralysis, foreign bodies in the bladder, sacculi, dilated bladder, tumours; sometimes abnormal conditions of the urine itself, and lastly it exists as a sequel of the acute malady. The symptoms are:—micturition increased in frequency, usually with some pain preceding the want to perform it; pains about the pelvis and perineum. The urine is more or less cloudy from the presence of an undue quantity of mucus from the lining membrane of the bladder. This is always present in all forms of chronic cystitis; without it indeed there is no inflammation. But the amount and the character of the mucous deposit differ greatly in different cases; thus, while in ordinary examples the quantity is inconsiderable, only producing cloudiness of the urine, in others it is so great as to form a large dense mass of semi-gelatinous material, giving rise to the term "catarrh of the bladder" as a mode of distinguishing it.

The medical treatment of chronic cystitis consists first in providing that the urine which enters the bladder from the kidneys, shall arrive in a state as little liable to irritate the viscus as possible. It should not be too concentrated, hence diluent drinks are usually beneficial · nor too acid, on which account liquor potassæ or citrate of potash in small but frequently repeated doses are often useful. The urine may be alkaline when it issues from the bladder from admixture of mucus or from ammonia produced by decomposed urea, although it has been secreted with the usual or even with an augmented degree of acidity, and if this be barely neutralised in the manner suggested, the inflamed bladder is less irritated and less inflammatory products are found than would otherwise be the case (Dr. G. Owen Rees). Then there are numerous infusions and other agents which appear to exert a salutary influence on the urinary tract of mucous membrane, when it is inflamed, which shall be briefly noticed. The infusions of buchu, uva ursi, parsley breakstone, alchimella arvensis and common hop, the decoctions of pareira brava, triticum repens, marsh mallow, linseed and pearl barley, are all useful, but should be administered in much larger doses than are usually given, of none giving less that ten ounces daily. Sometimes small doses of the balsams; such as sandal-wood oil, copaiba, or of compound tincture of benzoin, of benzoic acid, are useful; more commonly they merely derange the stomach and are without value. The action of the bladder is also tranquillised by sedatives of the nervous system, such as henbane, given in full doses, thirty to sixty minims frequently repeated; belladonna in moderate quantity, morphia in small doses. The tincture of the perchloride of iron has a reputation in some forms of the malady, and with justice.

Counter-irritation is a valuable part of the treatment; the best form perhaps being the application of a hot linseed poultice the surface of which is sprinkled with flour of mustard, to the sacral, suprapubic and perineal regions.

Injections of the bladder with warm water, or with very weak solutions of acetate of lead, of hydrochloric acid, of nitrate of silver, carbolic acid, biborate of soda and glycerine, quinine, &c., are of great value, but as they belong strictly to surgical treatment will not be further named here. They are especially applicable to those cases in which the mucous discharge is excessive.

I have already named *gout* as an undoubted, although rare, cause of cystitis. I have, still more rarely, witnessed an attack of gout on the bladder, without inflammation of that organ. A patient, whose family and personal antecedents are notably gouty, has sudden and periodical attacks of acute pain in the joints of the lower limbs, and sometimes also in the region of the bladder, the latter occurring simultaneously or alternately with the arthritic pain. There is general fever, furred tongue; the urine is soon charged with lithates, but inflammatory products are absent. The attack lasts a day or two, and the patient's sufferings are distressing while the bladder is affected; micturition being very frequent, and extremely painful. It is relieved by large doses of morphia, and ceases suddenly. The patient exhibiting such phenomena has, of course, no obstructive or other organic disease of the organs whatever, and is perfectly well in the interval of the attacks, which may occur once a year or so. The treatment consists, first, in hot hip-baths, with large doses of any opiate which best agrees with the patient, followed by free mercurial purgation, and afterwards by the regimen and management best adapted to the gouty patient.

Incontinence of Urine.—By this term is intended the habitual escape of urine without the knowledge, or against the will, of the patient, and without any power on his part to control the flow. In the adult, male or female, surgical examination is essential, in order to discover the cause. But the condition is very common in childhood, and is generally amenable to medical treatment. Usually it occurs only during sleep, although in a few instances the incontinence takes place when the child is conscious during the day. It is often almost congenital and continues, if not checked, even up to and beyond the time of puberty; frequently being the cause of great discomfort, and even of positive injury, by incapacitating the subject of it from school life, and other engagements. It occurs in children of very different temperaments, perhaps more commonly in those whose intellectual capacity is below the average than among others. The treatment consists, first, in permitting only small quantities of fluid for diet after the mid-day meal, in taking up the child after his first sleep when the elders retire, and making him empty his bladder then; in eliminating from his diet indigestible food, which might make him restless at night, and especially fermented liquors, if he has been accustomed to them; in promoting his general health by every means; ascertaining whether or no he is troubled with worms; if the urine be unduly acid, and the like. Secondly, by administering, before all things, belladonna. This agent has the power of paralysing the muscular fibres of the bladder; hence its bad effect in some cases of weak bladder in elderly men. By its means we can effectually antagonise the hyperactivity of the organ in these children; and, having paralysed it more or less during a period of two or three months, the habit of retention is acquired, and the incontinence does not reappear, or only to a slight extent, when the medicine has been discontinued. It is rare for belladonna to fail, if it be administered in sufficient doses, and its influence be maintained for a sufficiently long period. I give it thus: a child, say of eight years old, should commence with ten minims of the tincture in water, every afternoon, and at bed-time; after four or five days, the dose should be increased three or four minims, and so on, after similar terms, until it reaches at least thirty or forty minims twice a day. If no great inconvenience is experienced by this quantity, the dose may be again gradually and steadily increased, and a couple of months or so may pass before the maximum has been attained; and, if manifest improvement results, it should be continued, and finally gradually diminished, until one nightly dose only is required. I am never satisfied that it is desirable to employ any other agent until belladonna has had such a trial as this just described. That having been done, and without benefit, other remedies may be enumerated, as follows:—the addition of the tincture of the perchloride of iron, or steel wine, to the belladonna; the addition of strychnia, say $\frac{1}{50}$ of a grain, to each dose; for patients arriving at the age of puberty, the passing of a soft bougie every day, or alternate day, for a short time; the cauterization, five and ten grains of lunar caustic to the ounce of water, of the urethra and neck of the bladder; at any age the operation of circumcision, if there is a preternaturally long prepuce, or if congenital phymosis exists. For town-bred children, a wild life, during an entire summer, on Welsh or Scotch hills, pony-riding, and physical exercises of all kinds, with very little intellectual training, I have found successful in some notable cases, which had resisted all other treatment.

Retention of urine has been referred to above, and of course implies surgical treatment. *Atony*, and *over-distention of the bladder*, are among its causes in adult patients. This condition not unfrequently follows a prolonged voluntary retention by a patient who had been in circumstances in which he was unable to comply with pressing wants to micturate, and while regarding the symptoms met with in all these cases, it is never to be forgotten that *involuntary micturition indicates retention, and not incontinence.* There are a few exceptions to this rule, but very few. Hence, when the adult patient complains that urine escapes, either at night, or when making muscular efforts, without his ability altogether to control it, absolute necessity exists that his bladder should be explored by a catheter immediately after a voluntary act of micturition, in order to ascertain if any urine remains behind which by his own efforts he was unable to pass. That being so, to the extent of two or more ounces, as a rule the mechanical emptying of the organ every day is the essential treatment, if the patient is to regain the natural power, and maintain his urinary organs in a healthy state in the future.

Paralysis of the bladder gives rise to similar phenomena. By this term is not intended the incompetency already described, nor the inability to void urine which occurs from hypertrophied prostate, although the term is frequently applied, most unwisely, to both those conditions. Paralysis being logically only applicable to the result of lesions in the cerebro-spinal system, that incompetency on the part of the bladder, either to retain or to expel its contents [almost always the latter], which is due to such lesion, is alone so designated here.

No specific medical treatment, as regards the bladder, can be advised. In these circumstances a bladder which does not empty itself, must, before all things, be emptied by the catheter. Meanwhile the medical treatment of the central lesion will be determined by the entire group of symptoms, and the patient's general state, and will be pursued accordingly.

Neither can any medical treatment be suggested for any of those *organic changes* which affect the bladder, the existence of which may not be overlooked in diagnosing any urinary affection, and which may be enumerated as follows:—hypertrophy of the muscular coats of the bladder, most commonly resulting from prostatic obstruction, or stricture of the urethra; sacculation of the mucous membrane, occasioned by its being forced between the crossing fasciculi of the muscular coat, so that cavities are formed, capable of holding from one to fifteen, or even twenty, ounces of urine. The bladder is also the seat of villous growth, occasioning prolonged and considerable hæmorrhage; of epithelioma; of true cancerous formations; and, very rarely indeed, of fibrous polypoid growths.

For the significance of bleeding, from these and many other sources in the entire urinary system, a phenomenon broadly spoken of as Hæmaturia, the reader is referred to the article on that subject.

§ VII. DISEASES OF THE FEMALE REPRODUCTIVE ORGANS.

- A. Uterus.
    - Changes in Form and Position.
    - Disorders of Function.
    - Inflammation.
    - Growths.
- B. Periuterine Hæmatocele.
- C. Pelvic Cellulitis.
- D. Ovaries.
    - Inflammation.
    - Growths.

CHANGES IN THE SHAPE AND POSITION OF THE UTERUS.

By Graily Hewitt, M.D., F.R.C.P.

Introductory Remarks.—The system of uterine pathology which the author has advocated,[1] imputes to alterations in the shape of the uterus, a very prominent place, change of shape of the uterus being considered to have a very important effect in causing or intensifying certain other morbid conditions of the organ in question with which it is frequently associated.

These alterations in the shape of the uterus which have of late received renewed attention, and which have been further carefully studied by several observers of eminence, can no longer be placed in a subordinate position in any system of uterine pathology professing to be in conformity with the present state of knowledge. The clinical evidence of the close relation subsisting between these alterations of the shape of the uterus and the various troublesome symptoms and discomforts experienced by those who are the subjects of them, is too strong to be any longer disregarded. The uterus is undoubtedly liable to *functional* disease, and such functional disease may exist by itself. But it is also liable to what may be termed *mechanical* disease, and that to an extent which has not until of late years been suspected. Many of the cases of so-called functional disease, turn out on investigation to be cases of mechanical disease, and the question now, so often debated, is how much of the disorder present is functional, or how much merely mechanical. Thus amenorrhœa, menorrhagia, dysmenorrhœa, were, until lately, looked on as almost exclusively functional diseases, a view which can no longer be maintained. Again, the conditions hitherto denominated, "inflammation" and "congestion" of the uterus, and which have been regarded as substantial diseases, can be shown to be largely dependent on mechanical changes in the shape and position of the uterus. How much of the congestion or (so-called) inflammation is due to these mechanical changes is matter for important debate, and in point of fact, where differences of opinion prevail, it is principally as to the *primary* part played by the one or the other of these two elements in the case.

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[1] Diseases of Women, 3rd edit., 1872.

It is certain that *the condition of the tissues* of the uterus is matter in all cases for separate, careful consideration. The relation of these tissue changes to the mechanical diseases is one of the utmost practical importance, and in the author's view, this relation demands the most careful study and attention. Tissue changes themselves constitutional in nature, do in fact frequently cause, or predispose to various mechanical diseases of the uterus, and these mechanical diseases in their turn intensify the effects of the primary tissue changes. Further than this, the mechanical diseases themselves produce tissue changes peculiar and characteristic.

It is obvious enough that physical changes in the tissues of the uterus will be liable in a very important manner to predispose to the occurrence of such alterations as those we have now to consider and describe. Hitherto little attention has been bestowed on the etiology of uterine distortions, because the distortion has been looked upon as unimportant. Moreover, concurrently with the revival of attention to these distortions, the unsatisfactory character of the "inflammatory" theory of uterine disease has become more and more evident. The tissue changes in the uterus so long described as "inflammation," "chronic inflammation," &c., can be shown to be susceptible of less recondite and more intelligible explanation.

CONGENITAL DEFORMITY, OR DEFECTIVE DEVELOPMENT OF THE UTERUS.

Rudimentary formation.—Complete absence of the uterus is rare. Rudimentary formation is occasionally witnessed; the uterus may be the size of a pea or even smaller. Conjoined with this the vagina may be also rudimentary. The external organs may be apparently normal.

Infantile Uterus.—At puberty no further increase in size occurs. Conjoined with this the uterus may be abnormal in other particulars or not. The os may be imperforate. In a few cases further development occurs later on.

Uterus Unicornis.—The uterus is divided above into two cornua, one of which is well developed; the other small and rudimentary.

Double Uterus.—Of this there are several varieties. The separation may be entirely complete, even involving the vagina, each uterus having a separate vagina. The uterus may be externally apparently single, but completely divided into two internally by a septum, or this internal septum may only extend partly downwards. There are other variations. (*See* Kussmaul, *Von dem Mangel, der Verkümmerung und Verdopplung der Gebärmutter*, Würzburg, 1859).

DISTORTIONS OF THE UTERUS.

Distortions of the uterus include—*a.* Flexions; *b.* Inversion of the uterus partial or complete; *c.* Elongation.

When the uterus is bent forwards so that the uterine canal is curved, the concavity looking forwards, it is said to be *ante-flexed*. The reverse condition is *retroflexion*. In some cases there exists *lateriflexion*, together with some degree of ante- or retro-flexion.

Retroflexion as a distinct disease has been long recognised. But anteflexion has scarcely as yet, unless where the flexion is extreme in degree, been admitted to its proper rank as a definite and distinct affection. The reason for this omission is the circumstance that the uterus is normally slightly bent forwards, and the precise line of demarcation between normal and abnormal inclination forwards has not been accurately defined.

In general terms it may be stated that distortions of the uterus almost invariably give rise to discomforts or actual pains, which discomforts and pains are particularly produced by motion (uterine dyskinesia), and not unfrequently to such a degree that the patient is completely invalided, that they generally materially interfere with the due performance of the functions of the uterus, giving rise not seldom to dysmenorrhœa, or menorrhagia—often preventing conception and frequently occasioning abortion; that the various morbid conditions of the uterus, congestion, enlargement and so-called chronic inflammation of the organ are commonly associated with these distortions, and that the secretions of the mucous surfaces of the uterus are liable to be much increased and altered in character in cases where distortions are present. They, at the same time, give rise to various mechanical disturbances in the adjacent organs—the rectum and bladder—occasioning difficulty in defæcation, and in various ways disturbing the performance of the functions of the bladder.

Distortions of the uterus are of necessity associated with alterations of *position* of the organ—a common condition present is descent of the body of the uterus or fundus uteri in the pelvis. In cases of anteflexion, the descent of the fundus is to some extent limited by the bladder and other anterior connections of the uterus, and the dislocation of the fundus is not so considerable as in cases of retroflexion, when the fundus may be found to have descended almost to the rectal aperture.

ETIOLOGY.—Distortions of the uterus most frequently occur slowly, the degree of the distortion being at first slight, and increasing gradually; but they may originate in an acute form.

The *predisposing causes* deserve a careful attention. They include all circumstances which may weaken that inherent power which the healthy uterus possesses of resisting any attempt at alteration of its shape. The healthy uterus maintains its shape partly by the aid of its ligaments and general connections, but mainly, it may be said, by virtue of its own thickness and the resistance of its tissues. In point of fact, the heavier part of the uterus, that is to say, the fundus, has, practically speaking, no fixity, and were it not for the rigidity of the uterus itself would be readily bent backwards or forwards. The uterus in the healthy state has, as regards its centre, but little motion; the chief attachments of the organ surround it at this situation and prevent extensive movement. The centre of the uterus may be considered as an axis of rotation, the fundus being allowed to move backwards or forwards to some extent and the cervix uteri also participating in this motion. Probably the normal rotating motion of the uterus does not exceed a quarter of a circle, this being taken to represent the distance between extreme anteversion and retroversion. Further, in practising these evolutions it is to be understood that the uterus moves as a solid rigid body, and that it actually bends but little in the process. Probably a certain amount of bending-over actually occurs in individuals in a state of health during these normal movements of the fundus uteri backwards or forwards as the case may be, but then a rebound occurs and the organ returns to its original position and shape when the force producing the disturbance is no longer in operation.

Congestion of the Uterus.—Congestion of the uterus is a condition doubtless predisposing to the occurrence of flexions. But as will be presently shown (see p. 671) congestion of the uterus is properly speaking an effect rather than a cause of alteration in the shape of the organ. See also succeeding remarks on Mal-nutrition of the Uterus.

Mal-nutrition of the Uterus.—In common with other organs of the body, the uterus is liable to suffer from defective nutrition of its tissues. So well marked are the clinical features of cases of this kind, and so important are they in their various bearings, that it is deemed necessary to direct special attention to this subject.

This condition of the uterus is characterised by *unusual softness and want of firmness of the whole organ,* plainly recognisable by the touch so far as the cervix is concerned, and as regards the body of the uterus rendered evident by other facts of the case. The bulk of the uterus is less than usual, unless, as is not rarely the case, congestion be superadded. The softness of the tissues of the uterus may be so extreme that to the touch the vaginal portion feels almost as soft as in the latter months of pregnancy, instead of presenting the firm resistant feel of the healthy uterus.

Cases of malnutrition of the uterus present themselves in their most typical form in young women between the ages of sixteen and eighteen, and always in cases where there is abundant evidence of weakness and previous defective nutrition of the body generally. This condition may probably also be induced by the prostrating effects of severe fevers. The cases here alluded to include those described by Sir J. Y. Simpson as cases of super-involution of the uterus, but these latter cases occur in women who have borne one or more children.

The rapid development of the genital organs which occurs normally on the arrival of puberty demands a free and full supply of nourishment. In some rare instances, as is well known, the uterus never does attain its full development, the retardation probably being due to other causes; but this class of cases differs from those now under consideration. Here, we have the uterus fully developed and of the normal length, but its tissues are abnormally soft. From the circumstance that this is observed in association with great general weakness, the inference is obvious that the condition of the uterus is one of malnutrition. The clinical history of such cases and the results of treatment give the fullest confirmation of this view.

The undue softness of the uterine tissues reveals itself in the liability of the patient to suffer from flexion of the organ. Exertions of various kinds readily displace the uterus and distort its shape, and the suffering produced by this change of shape may be the first indication that the uterus is in any way affected. The uterus may retain its proper shape if circumstances are favourable, and with returning health and vigour of the system generally the uterus becomes also healthy. But undue, or even ordinary, exertions tell with unfavourable effect in many cases, and the softness of the uterine tissues paves the way to other disorders.

Young women presenting this condition feel a difficulty in walking more than a very short distance without discomfort (uterine dyskinesia). They suffer frequently from a feeling of nausea on rising in the morning, often persisting during the day or induced by exertion. They present various so-called "hysterical" symptoms. They are considered "delicate," perhaps whimsical, and the indisposition for exertion is not seldom regarded as a mental rather than a physical disturbance. The sympathetic effects on the stomach consist in anorexia, tendency to nausea or vomiting; the quantity of food taken daily becoming less and less, the patient eventually falls into a state of semi-starvation. The uterine malady is thus perpetuated, and the tissues of the organ lose still further their normal tone and firmness. The uterus fails to perform its functions properly, and dysmenorrhœa or amenorrhœa in various degrees of intensity may be observed. In a few cases severe menorrhagia occurs, of which the writer has seen two or three very notable instances.

Following the occurrence of this softness of the tissues of the uterus, in many instances there is observed later on a chronic, well-marked distortion of the shape of the uterus, with atrophy of the uterine wall at the centre of the organ, usually the part where the flexure occurs. But it is to be remarked that when the flexion is well established the softness may have quite disappeared. This point will be further considered later on.

Undue softness of the uterus, persisting only for a few weeks, may have no permanently injurious effect. But continuing for some months or longer, it rarely fails to lead to some such alteration in the shape of the organ, as to entail more or less permanent disability of various kinds. Locomotive power is apt to be most seriously interfered with.

Women who have had children, or who have been pregnant, may become of course the subjects of malnutrition of the uterus. In such cases the uterus is not only soft but it is generally larger than usual. Imperfect involution of the uterus is frequently due to defective nutrition of the organ. The tissues do not contract, the organ is large, soft, and unwieldy, and is very much at the mercy of external forces.

The great size and softness of the uterus render its distortion and displacement a matter of ease. Miscarriage after miscarriage is not seldom a consequence, the uterine walls appearing to have more or less completely lost their rigidity, and in some cases they may be so soft that it is even difficult to define by the touch the outline of the organ.

Clinical observation has led to the opinion here expressed that undue softness of the uterus is mainly due to malnutrition of the organ. The softness may be due either to feebleness and imperfect development of the muscular elements in the uterus, or to a deranged nervous action, or to deficient circulatory power and lessened blood supply, or it may be due to all these causes conjoined. It might be described as "atonicity" of the uterus but for the fact that the well-nourished uterus may fall into an atonic state; mere softness does not necessarily imply malnutrition, for the healthy uterus undoubtedly undergoes changes in regard to hardness and softness under certain circumstances. The dilatation of the cervix uteri by a tent, for example, produces a quite remarkable softness of the tissues of the os uteri. Further, contractions and relaxations of the uterus are now pretty generally admitted to occur during menstruation, especially in cases when dysmenorrhœa is observed, and under these circumstances alternate hardness and softness of the uterine tissues might be detected.

General weakness must be set down as a predisposing cause of uterine distortion, whether that weakness proceed from a general feebleness consequent on a long course of imperfect nutrition of the body, or be due to the effects of fevers or other prostrating diseases. Some of the most troublesome cases of uterine distortion have plainly their origin in this way; after some weeks or months of debilitating illness, from scarlet fever for instance, the patient begins to move about, and whether from the feeble resistance of the uterine tissues themselves, or the weakened condition of the uterine attachments, one or both, mechanical mischief results and uterine distortion is originated.

Sub-involution of the Uterus.—This is a most important condition in regard to the etiology of flexions of the uterus. The uterus increases considerably in weight and size during pregnancy. Thus when pregnancy is at an end, and the uterus is completely emptied of its contents, the organ is thicker, heavier, and very much larger than usual. Involution of the organ, as it is termed, follows, and in a few weeks the uterus regains its previous size. Such is the normal course. This course is frequently disturbed, the uterus failing to undergo its proper reduction in size. The term sub-involution, first employed by the late Sir J. Y. Simpson, is now generally employed to designate such cases.

Sub-involution of the uterus is a frequent concomitant of abortion, as well as of deliveries at full terms. The causes of sub-involution of the uterus are general and local. The *general* causes are *great debility*, in consequence of which the nutritive changes which should proceed rapidly in the uterus are retarded or impeded. Weakly women are peculiarly prone to suffer from sub-involution. Any *general* or *constitutional disease* may give rise to it. The *local* causes are frequently mechanical in their action. Thus the normal involution is often retarded by alterations in the shape of the uterus, resulting from strains and undue exertion too quickly following labour. The distortion impedes the circulation and the nutrition process is disturbed. The sub-involution is not uncommonly the primary cause of the alteration in shape, as when a patient six weeks after her labour, with the uterus in a state of sub-involution due to general debility, takes a long walk, produces thereby a distortion in the shape of the uterus, and two or three months later, perhaps, is discovered to have a large retroflexed or anteflexed uterus. Here the sub-involution has a primary as well as a secondary cause.

In the list of local causes must be mentioned previous distortion of the uterus, for a long standing distortion frequently recurs after pregnancy is over, and such recurrence may act very powerfully in retarding uterine involution.

Puerperal fever, now more correctly designated puerperal septicæmia, may produce sub-involution of the uterus. Observation has convinced the author that involution is at once arrested by the existence of puerperal fever, one of the first effects being apparently a paralysis of the muscular fibres.

Sub-involution of the uterus is, not unfrequently, either directly or indirectly, the cause of other diseases of the uterus; many troublesome cases of menorrhagia and leucorrhœa have their origin therein. Prolapsus and change of form of the uterus are frequent effects. Chronic hypertrophy of the uterus is the result in many instances. Disorders of micturition and defæcation may also result from the increased weight and bulk of the organ.

Congestion of the uterus is frequently associated with sub-involution. In fact both conditions are frequently traceable to the same cause—general debility. And when distortion is superadded to sub-involution, the congestion is liable to be of an extreme character.

Laceration of the perinæum is a most important predisposing cause of uterine flexion and displacement. The floor of the vagina is thus diminished in area and thickness. Perineal deficiency most frequently tends to give rise to the anterior descent of the fundus uteri; the bladder first descends, and this is followed by the uterus. The tendency is thus to the production of anteversion and anteflexion, and severe degrees of anteflexion coupled with cystocele are sometimes met with in cases of extensive injury to the perinæum, the result of labour. More rarely retroflexion and perineal deficiency are found associated.

An important predisposing cause, is alteration of the position of the uterus as a whole. The uterus as a whole has certain normal movements irrespective of alterations in the direction of its axis, or of alterations in its degree of curvature, viz. movement forwards or backwards, and upwards and downwards. And there can be no doubt, that when the normal movements in these directions are exceeded, this circumstance favours a change of shape of the organ. Abnormal mobility of the uterus thus constitutes a predisposition to change of the shape of the organ. Pregnancy is apt to leave behind it, especially in weakly individuals, an abnormal loosening of the general connections of the uterus. Then the uterus falls as a whole too low, or moves too far forwards or backwards, and when thus removed from its proper position in the pelvis, it is more liable to be bent, should a force capable of causing a bending be brought into play.

Exciting causes.—Decided flexion of the uterus may be produced in what may be termed an *acute* way. Various accidents, principally those which give rise to a sudden jar or shake of the whole trunk of the body, may give rise to it. The following are instances :—A young lady is rolled over and over in a railway carriage down an embankment; a young woman in a spirit of bravado carries a heavy weight across the room; or, while standing at a horse's head, the animal suddenly breaks away and drags her along the ground; or coming down stairs the foot slips and she slides down several steps, the lower part of the spine being brought into violent collision with the edges of the steps; or walking in the streets she suddenly slips upon a piece of orange-peel; or unaccustomed to horse exercise, she rides on a hard-mouthed spirited horse; or walks for several hours consecutively in a mountainous country, having been unaccustomed previously to take long walks; or at a gymnasium she practises jumping down from one elevation to a considerably lower one. These are actual instances of acutely and suddenly produced distortion of the uterus occurring in tolerably healthy and previously sound individuals, who became thereupon completely laid up and incapacitated from the event in question. Many other similar histories might be quoted.

But it frequently happens that the distortion originates in a less acute manner, and in consequence of the continued operation of a less powerful exciting cause. Of these cases the following may be cited as instances :—

A young woman not particularly strong, engages in an occupation requiring prolonged standing, or too frequent lifting of heavy bodies. A young lady at school, and not sufficiently fed, is made to take long walks. A patient recently recovered from a long illness, is allowed to take too vigorous exercise. Numberless instances of this kind might be quoted, but they resolve themselves usually into a combination of the two conditions: (1) a rather undue weakness of the uterus or its supports, and (2) a slight, but continually acting force compressing the pelvic viscera from above, and having the result that, more or less speedily, the fundus uteri is pushed forwards or backwards, or laterally as well as downwards, and thus distortion is produced.

And it is not very uncommon to meet with very considerable and serious flexions, ante, retro, or lateral, in patients who have never suffered from any severe physical shock or accident. In such cases, the tendency is to assume that the condition is congenital, but a considerable congenital flexion appears really to be one of the very rarest of events, and the explanation of the condition will usually be found if a careful investigation of the previous history of the patient be made.

Here it seems proper to state the result of the author's observations, that certain physical exertions would be wisely avoided by all but very robust young women, such as riding on horseback, taking long walks (over an hour at a time being considered "long"), violent gymnastic exercises, &c. And clinical facts show that young women previously in robust health, are liable to be seriously damaged by such exercises.

Complications and effects.—It will be convenient to consider the complications and effects of flexion of the uterus under one head, inasmuch as opinions would be divided as to the proper use of the term "complication" or "effect" in certain instances. One important complication is the change in the *position* of the uterus. Flexion of the uterus of course implies a certain change in the position of the fundus uteri. The uterus does not undergo a change of position *as a whole* in consequence of such flexion. And *vice versâ*, the uterus may have its position very much changed, quite irrespective of the occurrence of flexion. As a matter of fact considerable degrees of flexion are generally associated with very considerable degrees of lowering of the uterus, and descent of the whole organ towards the floor and outlet of the pelvis. This subject, which is one of very great importance, will be found discussed separately further on.

Congestion of the Uterus.—The relations of congestion of the uterus to flexions of the organ are most important. Congestion may be both a cause and an effect of flexion of the uterus. It will be convenient in this place to consider congestion of the uterus from a broad point of view, and to explain the general relation subsisting between this condition and the various distortions of the uterus.

Undue fulness of the blood-vessels of the uterus is a condition frequently met with. The uterus is a very vascular organ, its texture, notably as regards the body of the uterus, although firm, might be almost described as spongy, and the interspaces, so evident on section to the naked eye, are susceptible of considerable distention with blood. In fact the structure of the organ, and its extensive blood supply, predispose it to the occurrence of congestion.

General congestion of the uterus is probably present to a certain extent during menstruation, as first insisted on by Rouget, the organ being then fuller, heavier, and more bulky than usual. It does not follow from this that menstruation is caused by the congestion thus occurring. Dr. John Williams, who has recently devoted much attention to this subject, considers, indeed, that menstruation is not in any way dependent on congestion of the uterus. Congestion of the uterus is probably also present during sexual congress. Congestion of the uterus in an acute form, probably occurs in cases where menstruation is suppressed from external application of cold, standing in wet, &c., possibly also from emotional disturbing influences. In a more gradual, slowly induced manner, general congestion of the uterus may be produced by an immense variety of causes. In all cases of general weakness or debility, when the blood current is slow and the circulation feeble, in cases of disease of the heart, of the liver, of the digestive organs, in cases of great debility following attacks of fever of various kinds, in cases when the uterus itself has undergone considerable draining of blood from its interior,—in all these cases chronic general congestion of the uterus may be present to a marked degree.

Sub-involution of the uterus following on child-birth or miscarriage is very frequently associated with congestion of the organ, and in fact in many cases the congestion is the principal and prominent condition then observed.

Considerable increase in the bulk and weight of the uterus are necessarily accompaniments of its congestion. Very important dynamic results may ensue from this increase in size and weight, for it so happens that the body of the uterus, which is the part of the organ most affected by such congestion, increase of size, &c., is the part least capable of preserving its proper position and relations under unfavourable circumstances.

Coupled with this general congestion of the uterus we find frequently undue softness of the tissues. Not always so, however, for when the congestion has lasted a long time the uterus may become harder than usual; the congestion is exchanged for hardness and increase of size. The previous remarks apply to the general causes of congestion of the uterus. But it appears that in a very considerable number of cases congestion of the uterus is due to a change in the shape of the organ. The explanation of this occurrence is sufficiently simple. The blood-vessels of the uterus, for the most part, enter and leave it about the centre of the organ, passing from this central situation upwards to the fundus and downwards to the cervix. The two extremities of the uterus are, moreover, comparatively free and unattached. Any compression of the central part of the uterus, that is to say, at or near the situation of the internal os uteri where the body of the uterus joins the cervix, is thus attended necessarily with an obstruction to the flow of blood to and from the cervix uteri at its lowest part. Hence, when the uterus becomes bent, an obstruction in the circulation in the organ may occur, and congestion ensues at one or both extremities of the uterus. This mechanically produced congestion may affect the fundus exclusively or the vaginal portion of the uterus exclusively, according to the precise seat of the flexion. Some few years ago, the author described the pathological congestion so occurring as "strangulation" of the uterus. Klob, had, it appears, previously, though this was then unknown to the present writer, alluded to the influence of flexions in producing congestion of the uterus. Dr. Gaillard Thomas, also considers this as an important etiological element. The effect of an acute flexion of the uterus is, indeed, so far as the circulation is concerned, nearly the same as would be produced if a ligature were placed round the uterus at its middle, not sufficiently tight to close the arterial, but enough to materially retard the return through the venous current. A due recognition of the influence of flexion of the uterus in thus producing congestion, is essential to a complete understanding of the subject.

A general result of congestion of the uterus is *increase in size and weight*, due, at first, to the increased quantity of blood in the tissues, but, in chronic cases, to actual increase in the quantity of solid constituents. Various terms are employed to designate this condition. It is essentially an hypertrophy. The term "chronic inflammation" conveys a wrong impression, and is discarded by several writers of eminence. Dr. Gaillard Thomas terms it "Areolar hyperplasia" "Congestive hypertrophy," would, having regard to its etiology, perhaps more correctly designate the condition in question.

In cases of "congestive hypertrophy," the tissues are at first softer than usual. They end often by becoming very much harder than usual, and when the organ has become altered in shape, as is not uncommonly the case, we have a combination of extreme hardness with distortion of shape and increase of size, the increase in size—congestive hypertrophy—affecting the fundus alone, the cervix alone, or both conjointly.

Congestive hypertrophy of the cervix, when giving rise to much swelling of this part produces a very open condition of the os uteri externum. The surface of the os uteri may also be extremely vascular, and present those various appearances formerly described as "ulcerations." It is well known that these so-called ulcerations attracted at one time well nigh the whole attention of uterine pathologists. The extreme vascularity of the mucous membrane lining the cervix and the abrupt line of demarcation presented between this and the adjacent mucous membrane covering the lips of the os uteri, appear to have given a false impression, the abrupt line of division having been probably taken to be the edge of an ulcer in many cases. The natural shape of the os uteri is rarely preserved in a long standing case of congestive hypertrophy of the uterus.

Congestive hypertrophy in its early stage—that of softness—is often associated with mal-nutrition of the organ (see *ante*, p. 666). It is not seldom a sequel of defective involution after labour.

Congestion of the uterus may be *acute* or *chronic*. An intense form of acute congestion is witnessed in severe flexion of the uterus. The more severe forms of chronic congestion are observed in association with defective involution following delivery. An acute attack has a tendency to become chronic, and the chronic form is liable at any moment to become acute.

Probably a useful distinction might be drawn between *active* and *passive* congestion of the uterus. Thus, active congestion might follow a chill; passive congestion would result from a flexion of the uterus. These may be mentioned as typical events.

Disturbances of Functions of the Uterus.—A very common effect of flexion of the uterus is obstruction to the catamenial flow. Many of the most severe cases of *dysmenorrhœa* are directly due to the constriction of the uterine canal at or near the internal os uteri which the flexion produces.

A further not very uncommon effect is retention of uterine discharges within the cavity of the uterus. Puriform or semi-purulent fluids retained in the uterus escape from time to time in gushes, sometimes accompanied by pains not unlike dysmenorrhœal pains. Endo-metritis is the natural result of this condition, and the uterine cavity becomes not only irritated, but distended by the retained fluid.

Leucorrhœa, due to increased cervical secretion, is common in cases where flexion *plus* congestion exists.

Menorrhagia is a rather frequent accompaniment. It is partly due to the obstructed flow causing accumulation in the uterus and distension of that cavity, and partly to the unusual congestion present. The loss of blood arising from flexion is sometimes very great.

Increase in the size of the uterus is a further effect observable in chronic cases. The two extremities of the uterus—the fundus and the vaginal portion of the cervix—more commonly exhibit this effect. A large hypertrophoid nodular condition of the vaginal cervical structures is witnessed in some long-standing cases. And the body of the uterus is similarly hypertrophied still more frequently.

Atrophy of the uterus is sometimes observed. More commonly, however, the atrophy is localized, and affects that part of the uterus only at the seat of the bend. It would appear that the thickness of the cervical canal is sometimes so materially reduced by the long continued pressure to which the tissues have been subjected that the wall is only the thickness of paper. This is liable to occur particularly on the concave side of the bend. But this atrophy is not a constant effect, nor is it constantly present on the concave side. As a matter of fact the thickness of the wall is sometimes even increased at this point, and the author has had occasion to observe an actual bulging at this situation on examining the outline of the uterus digitally. This local atrophy is a very serious obstacle to a perfectly successful curative treatment in long-standing cases.

The painful sensations and alterations of sensibility due to flexions are numerous and highly important; but they will be more appropriately described under the head of "Symptoms."

GENERAL REMARKS ON TREATMENT OF FLEXIONS OF THE UTERUS.—Here it will be well to point out the general principles on which the treatment of cases of flexion of the uterus should be conducted. More especially it is necessary to direct attention to those measures by which the various disordered conditions of the tissues of the uterus, which play so important a part in the etiology of flexions, may be remedied and prevented. Special appropriate treatment is required for various cases, but the general treatment is of exceeding importance, and mere mechanical treatment will lead but to poor results, unless due attention be paid to the amelioration and improvement of the general condition of the patient, by which latter means alone we can hope to satisfactorily influence the nutritional changes going on in the uterine tissues.

When softness, due to *mal-nutrition of the uterus*, is present, as is very frequently the case, the remedy is obvious. Food is required. Great difficulty may be experienced in administering it owing to the nausea, feebleness of stomach, and other causes. For such patients abundance of fresh air is very necessary, but the recumbent position during the greater part of the day is almost essential. A gentle walk may be allowed in some cases, but all violent exercises, such as riding on horseback and carriage exercise over rough roads, must be forbidden. It is found that patients eat best who follow this rule. The sympathetic nausea is thus prevented. Animal food given frequently, in small quantities, at first in form of soups or essences, afterwards in the solid state, succeeds best in restoring the firmness to the uterus. But many months may be required to effect the object. Baths, frictions, tonics of various kinds, change of air; whatever, in fact, tends to help the restoration of the general health will be serviceable.

In cases where *sub-involution of the uterus* is present, means must be adopted to hasten and promote the contraction of the uterus. When the form of the uterus is unaltered, ergot, tonics, and a thoroughly nutritious dietary will be found effectual. When the uterus is altered in shape mechanical treatment may be required, and is indeed imperative in some cases. The occasional straightening of the uterus by means of the sound, associated with maintenance of the dorsal position in ante-flexion cases, or the semi-prone position in cases of retro-flexion, will powerfully assist in the restoration of the uterus to its proper size and shape. Pessaries are requisite in some instances, and when the distortion and associated sub-involution are of long standing, they are indispensable. Whatever tends to promote healthy circulation in the uterus acts favourably in reducing its size. Warm injections containing Kreuznach salt in solution are beneficial.

The general Treatment of cases where Congestion is present.—Attention to the general health is of the utmost importance. When the uterine congestion arises from a general sluggish circulation, or from a general mal-nutrition, or from a special uterine mal-nutrition; under these circumstances no progress can be made unless the greatest care be taken to strengthen the patient. Much rest, fresh air, highly nourishing food, baths, frictions of the skin, change of scene, ferruginous tonics—these are powerful remedies if they are judiciously applied. In some cases the fundamental general weakness is such that a year or two of such treatment is required to restore to the patient an average strength. These are cases requiring emphatically a "bracing" system, as it is termed. But this bracing system must be applied with discrimination. Such feeble ill-nourished patients are not fitted to undertake long walks, or to ride on horseback, or to get up early in the morning, or to sit in the ordinary position for any length of time. Mischief of an almost irreparable kind is not seldom done by want of consideration in recommending some of these exercises and supposed aids to the recovery of health. The constitution and peculiarities of women are such that these vigorous procedures are not tolerated, and a chronic distortion of the uterus is liable to be the result of such injudicious recommendations. The amount of exercise should be limited, but the patient should be as much as possible in the open air.

Aperients are more or less necessary, for absence of exercise tends to produce constipation and inaction of the liver. Castor-oil taken in doses of a tea-spoonful daily is a valuable medicine. Enemata, daily, are better suited to some cases. Some of the foreign mineral waters, as Friedrichshall or Pullna, &c., are often useful. And it is well to give occasionally a moderately strong dose of some cholagogue medicine. Even small doses of blue pill are required in certain instances. Constipation must at all costs be prevented.

Rest.—One of the most powerful agents in the general treatment of flexion cases is "rest." This has been long known and acted on to a great extent. The horizontal position gives rest to the body generally, and also to the uterus, and when—as is most commonly the case—it is a very important object to prevent the further downward movement of the fundus uteri, the horizontal position is a great assistance. For cases of simple congestion, for cases of general or local mal-nutrition, when the uterus has not yet become decidedly distorted, this kind of rest for the uterus may be all that is required.

ANTEFLEXION OF THE UTERUS.—The uterus has normally a slight anterior concavity as regards its canal. The degree of the normal bend forwards has been the subject of much controversy. It appears that it is more pronounced just before puberty than afterwards, and that the normal healthy development of the uterus involves a considerable straightening of the canal. It follows that if circumstances retard this healthy development, the anterior bend will continue to be too pronounced after the age of puberty has been reached.

Anteflexion of the uterus exists in all degrees. In slight cases the two canals of the cervix and of the body of the uterus would be represented by two lines meeting at an angle of more than 90°. When severe the angle may be considerably less than a right angle, the organ being literally doubled upon itself. The curve actually formed is generally a parabola, or approaches thereto more or less closely.

Associated with anteflexion, there is usually a more or less considerable degree of anteversion. The os uteri approaches the centre of the sacrum while the fundus approaches the triangular ligament. There are infinite varieties in this respect. Simple extreme anteversion is rare. Further there is in many cases another very important change, viz., descent of the uterus as a whole, the organ presenting, not rarely, the three conditions, descent, anteversion, and acute anteflexion. Until these several elements are recognised, and their several degrees ascertained, the diagnosis cannot be said to be complete.

There is one peculiar variety of anteflexion, which consists in a simultaneous acute anteflexion with descent of the organ, and retroversion instead of anteversion. In this latter case the vaginal portion of the cervix points directly upwards and forwards; cases of this kind are extremely difficult to deal with successfully. Decided anteflexion with the uterus, as a whole, in its normal position is rare, but down it is sometimes observed.

Anteflexion of the uterus is a very common affection. In slight cases the real nature of the malady frequently escapes detection. In very severe cases it is not uncommonly overlooked, because no attempt is made to define the position and shape of the uterus by careful examination, or the presence of "displacement" being known, the importance of the distortion really present is underrated, the patient's symptoms and discomforts are not attributed to their real source, and a particular investigation of the case in this direction is never really undertaken. The principal explanation of this latter occurrence is the prevalence of what the author believes to be a completely mistaken idea as to anteflexion being a normal condition of the uterus. The normal anterior curvature is really very slight, and it is not accompanied with a descent of the uterus forwards and downwards, which in a greater or less degree characterizes this disease —for disease it most assuredly is.

It may be convenient to divide cases according to the degree of the flexion, into first, second, and third degrees. In the first degree the flexion is slight, in the third it is highly marked. In cases of anteflexion of the third degree, the body of the uterus is often not so low in the pelvis as in cases of the second degree.

In cases of anteflexion the uterus may be found soft and pliable from mal-nutrition, or large and soft, and pliable, from defective involution following abortion or delivery at term. Or it may be hard, the hardness being coupled with considerable increase of size, or with no alterations in this respect. There may be considerable congestion of the whole organ, or that congestion may be limited to the body of the uterus.

In women who have borne children, considerable anteflexion and anteversion are sometimes coupled with deficient perineal support, the perineum having been considerably lacerated during parturition. There may be also in such cases some degree of prolapsus of the bladder. The injury to the perineum in such instances, is the direct cause of the uterine displacement and distortion.

Symptoms.—These are not always characteristic as between this affection and other varieties of uterine distortion and displacement, but they are frequently so. *Frequency of micturition* is very commonly observed, constituting sometimes a symptom most troublesome to the patient. In some cases it almost amounts to incontinence. This symptom arises partly from the uterus actually occupying the space in the pelvis devoted to the bladder, and partly from the actual irritation of the pressure of the uterus on the floor of the bladder. Sometimes it is the result of an actual cystitis set up by the pressure of the uterus and the general irritation it produces. Retention of urine due to anteflexion is not common. *Pain,* referred to the groin on one or other side, and *pain in the back,* are common symptoms. This pain is exceedingly variable in regard to its degree. It is not a constant pain, and frequently hardly amounts to pain. It is produced by certain exertions, or intensified by them: stooping, lifting, going up and down stairs, taking a long walk; these may any of them give rise to marked discomfort. Not uncommonly the patient describes it as a bearing-down sensation, and she frequently very exactly and vividly describes the painful dragging, pushing sensations which would be expected to be experienced under such circumstances.

Sitting, especially sitting forwards, is generally painful, though some patients find a kind of relief in this position when the malady is of long standing. Locomotion is a trouble, but patients can frequently walk a moderate distance; not unfrequently, however, walking is a real impossibility. In acute cases motion is not attempted, or if it be practised it is at the cost of great aggravation of the existing pain. Many patients are entirely incapacitated for this reason. They walk less and less, or perhaps, encouraged by their friends to persevere in the attempt to walk, a complete break-down ensues, and the patient becomes a chronic invalid.

Locomotive inability (uterine dyskinesia) is one of the marked symptoms of anteflexion of the uterus, but it is not characteristic of it, for other displacements of the uterus may give rise to this symptom in an equally well-marked degree.

Nausea, or nausea and sickness, are symptoms of very great importance. They are rarely absent. The nausea may be slight or severe. It is mostly observed on first rising in the morning, but frequently it follows any exertion. The mere sitting at table often induces it. In very severe cases it is itself a real disease, imperilling the very existence of the patient; and cases are now and then seen in which the long continuance of the malady has brought about a near approach to starvation—wasting and extreme feebleness from want of food. Clinical observation plainly shows that the nausea is increased by whatever increases the descent of the fundus uteri, and thus exaggerates the existing flexion. This symptom is not peculiar to anteflexion, but may exist in other varieties of uterine distortion.

Constipation frequently exists in cases of anteflexion, and it is not uncommon to find that very strong medicines are required to procure action of the bowels. The difficulty more particularly occurs when the uterus is a good deal anteverted as well as flexed, and it appears to be produced by the forcible pressure of the cervix of the uterus against the floor of the pelvis, and on the rectum.

Swelling of the hypogastric region, usually limited to one side, is a rather constant symptom. It is a tympanitic distension, slight in amount, but it constitutes a discomfort of which the patient very much complains. It is absent in the early part of the day, but appears later on. Many patients believe themselves to have an actual tumour at this situation. This tumefaction has been considered by many observers to be dependent on ovarian irritation, and it is not usually ascribed to the cause here indicated. However, observation completely bears out the truth of the statement. It is difficult to give a quite satisfactory explanation of the rationale of the occurrence of this swelling, but that it occurs in connection with uterine flexion, and particularly in connection with anteflexion, is undoubted. It is always moreover intensified or produced by such mechanical movements as would be likely to increase temporarily the existing distortion of the uterus, such as standing or walking, or sitting upright.

Hysterical phenomena are in numberless instances associated with anteflexion. It cannot be said that their presence is in any way characteristic of this particular flexion of the uterus, for hysterical phenomena are or may be observable also in cases of retroflexion. The relation of hysteria to uterine flexions will be considered later on.

Diagnosis.—A careful digital examination is necessary in order to establish the diagnosis. An examination can be made per rectum, and the position and outline of the uterus may thus be tolerably accurately made out in cases where a vaginal examination is not judged to be imperative, or where it is objectionable. This method of examination is of the greatest service in making what may be termed an approximate diagnosis. In cases where no objection to a vaginal examination exists, the use of the finger aided by the sound enables us to make a very exact diagnosis of the case. In cases of slight anteflexion the finger must be pressed upwards through the vaginal roof, in order to define the outline of the body of the uterus. Normally the finger should encounter no resistance in this position. In severe cases of anteflexion the fundus can be felt and its breadth defined with the utmost readiness, and when there is also much anteversion the whole organ is found close to the floor of the pelvis. The size of the uterus is thus also accurately appreciated.

Cases of anteflexion may be confounded with fibroid tumour in the anterior wall of the uterus, a condition which may closely simulate it; the converse proposition equally holds good. Many cases of supposed fibroid tumour in the anterior wall are resolved by the use of the sound into cases of acute chronic anteflexion. When the flexion is not associated with anteversion the recognition of its existence is not easy. Unusual density of the cellular tissue above the roof of the vagina may hinder recognition of the outline of the uterus. And it must be borne in mind that the fundus is not as a rule quite in the middle line; there is frequently indeed a quite decided inclination to one side, although this does not, except in very rare instances, amount to lateriflexion. Lastly, the very peculiar cases of acute anteflexion, combined with retroversion, occasion some embarrassment in their diagnosis, for the fundus is of course not to be felt at all in front. The sound will be the means of resolving the doubt. The combined method of examination by the rectum and over the hypogastric region is often useful in the diagnosis of a doubtful case.

Prognosis and Treatment.—When the distortion is recent it is not difficult to cure. When it has become chronic, and the disorder is perhaps of some years' standing, the cure is not easy. The difficulty in chronic cases is very frequently enormously increased by the circumstance that the patient is profoundly affected with another disease, viz., general mal-nutrition. It is hopeless to endeavour to effect permanent alteration of the shape of the uterus until the general health is restored. All chronic cases are not of this character, for in some the patients have got over the general feebleness which really gave rise to the disease originally, and the uterus is hard and compact and very firmly fixed in its distorted condition. A chronic neurosis is established, and prolonged mechanical treatment may be required to unbend the uterus and to give it a more natural shape. There are cases of this latter variety which are almost impossible to cure completely in consequence of the tenuity of the uterine wall at the seat of the flexion. When the uterus has become much hypertrophied also this additional bulk is difficult to get rid of, and yet unless the weight of the fundus be lessened the disease will return after an apparently successful treatment.

In cases where the anteflexion is of recent occurrence, the flexion not severe, and the uterus as a whole not very low down in the pelvis, treatment such as the following may be sufficient:—The patient must be ordered to lie down as much as possible; the sitting position must be studiously avoided; lifting, carrying, stooping, and muscular exertions generally to be forbidden. The general health may flag if the patient is kept indoors; but carriage exercise is injurious unless taken in a semi-reclining position, and a short walk is less prejudicial than a ride in a carriage over a rough road. Persistence in this kind of mechanical treatment—which it really is—is often efficacious. Care should be taken that the bowels be opened daily without straining, and means taken to produce this result. Sea-baths, sponging with warm sea-water, tonics, fresh air, good food—these are adjuvants in the treatment not to be neglected.

If, as is not seldom the case, the general nutrition is greatly lowered, all treatment will be useless until this fundamental defect be remedied.

When the case is of long standing such treatment as that described above will prove merely palliative. In the case of young or unmarried women we have before us a choice of evils. If no direct local treatment be applied the patient may lapse into a permanent invalid, and her whole future prospects are perhaps endangered, or she has to submit to a course of treatment which is naturally distasteful. Another view may be perhaps taken of some of these chronic cases, viz., that the patient may after a while lose the principal of her discomforts, and that she may become habituated to the malady. It may be difficult to decide abstractedly, but in actual practice the circumstances of the case are usually a reliable guide. Assuredly there are numerous cases which it is simply cruel to allow to drag on from year to year unrelieved simply because the patient being unmarried local treatment is thought objectionable. This extreme is to be reprehended equally with the other extreme of applying local mechanical treatment in all cases of anteflexion indiscriminately. For married women affected with severe and long standing anteflexions, local measures are almost necessarily employed.

There are various methods of treating anteflexion of the uterus when local measures are thought advisable. Periodical straightening of the uterine canal by means of the sound associated with maintenance of the dorsal position answers very well up to a certain point. The employment of the stem pessary is from many points of view satisfactory, and in good hands safe; but it is difficult to manage, and is certainly not a plan to be employed indiscriminately. Permanently applied pressure upwards through the vaginal roof offers another, and, for many reasons, a more generally applicable method of treatment, and it may be combined with occasional use of the sound. The author's "cradle" pessary, in some one of its various modifications, and carefully adapted to the peculiarities of the case, offers great assistance in dealing with cases of anteflexion, whether recent or of long standing; but care must be taken that the pressure is made sufficiently high up and above the point at which the bend occurs.[1] When the uterine tissues have become very hard and firm this kind of pressure, alone, is of little avail in remedying the distortion. A frequently repeated use of the sound, and bending the uterus the reverse way, is then necessary. In some cases the stem pessary alone will be efficacious. In certain cases, when other mechanical supports are not tolerated, a small perfectly round air-ball pessary, 1¾ inches in diameter, will be found extremely useful.

Cases are now and then met with in which the anteflexion has established a permanent neurosis, and at the most compressed part acute tenderness is found to be present. These very severe cases, a few of which the author has met with in practice, are particularly troublesome to deal with. But they are extremely instructive as throwing considerable light on the cause of the pain usually felt in cases of flexion of the uterus. In these particular instances the tenderness was limited to precisely one spot; whenever the sound passed a particular part of the uterine canal acute pain was felt. The condensation and compression of the tissues implicating the nerves of the part appeared to be the cause of the pain, and acting on this view of the matter bi-lateral incision of the canal at this situation was in some of these cases performed. Subsequently the canal was maintained in a dilated state by a stem pessary for a few weeks. The treatment was successful in removing this abiding pain.

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[1] The plate published in the author's work, 3rd edit. 1872, representing the instrument *in situ*, is not a representation of the mode of applying it which he has now, for some time past, adopted. In the plate in question the pessary is shown pressing on the uterus too low down.

The question as to the necessity for cutting operations in the treatment of chronic anteflexions is one of some importance, for there is no doubt that a bi-lateral incision of the canal in some cases is of benefit and an assistance in carrying out the straightening treatment. Hitherto this operation has been principally done with a view to cure the supposed stricture at the internal os, or lower down the canal, and very little has been said of the acute flexion, which usually exists in these very cases. This flexion has been either entirely overlooked or thought of no consequence. Hence the cutting operation has had a comparatively small success, the effects of the operation passing off in a few weeks, and the uterus left as impervious as ever. Those cases only have given satisfaction when this cutting operation has been followed by a continuous dilatation treatment for some time. In fact the mere cutting is useless without this subsequent treatment. In the neurotic cases above alluded to the incision was valuable and useful; but it does not appear that, as a rule, it is a necessary procedure. The canal may be dilated with tents, and when sufficiently enlarged a stem pessary may be worn to maintain that degree of patency, and allowed to remain long enough for the uterine tissues to grow around it, and become consolidated in the new shape.

The element of time is of much importance in the treatment of chronic anteflexion. The cure is always tedious, and patience is required. Anteflexions are permanently improved by pregnancy for obvious reasons, but when the pregnancy is over the recumbent position should be maintained longer than usual.

The treatment of anteflexion in connection with pregnancy is a matter of great importance. When patients who have had anteflexion become pregnant, miscarriage is always to be feared. The absolute maintenance of the horizontal position during the first half of pregnancy is imperative in some cases. The round air-ball pessary is of great service at this juncture, a well-fitted cradle pessary will be found still more advantageous. These artificial supports are usually not required after the middle of pregnancy. The very close connection which subsists between anteflexion, and the liability to miscarriages, has not yet attracted professional attention, but according to the author's experience, this particular distortion of the uterus is by far the most common cause of early expulsion of the ovum.

Retroflexion of the Uterus.—Posterior displacements are not of such frequent occurrence as anterior displacements of the uterus. Retroversion unaccompanied by flexion is exceedingly rare. In all cases of retroflexion there is of course version more or less marked.

There are many varieties:—

1. In regard to the *degree* of the flexion. Thus in the *first* degree, where the organ is slightly bent backwards; in the *second* where it is more decidedly flexed, and in the *third* where the fundus is so much inclined backwards that the organ resembles half of a circle, the two extremities being very close to each other. Naturally, the more decided the flexion the lower the uterus as a whole. Thus in the third degree the fundus is generally very close to the anal aperture. 2. In regard to the *condition of the tissues* of the uterus, which may be very soft, or very hard, much swollen and congested, or comparatively normal in condition. In the early stage softness is usually present; in chronic cases the uterus may have become very hard. 3. In regard to the *shape* of the uterus. Thus in some cases the fundus, repeatedly thrust downwards, in the act of defæcation, and invaginated into the rectum, assumes a conical shape from its being moulded and compressed by the sphincter recti. In many cases the lips of the os uteri become knotty and turgid, especially the posterior lip, and finally the os is surrounded by hypertrophied hard lips. 4. In regard to the *canal* of the uterus, which in many cases is much narrowed. 5. In regard to the *cavity,* which is frequently much enlarged and distended by retained menstrual or mucous secretions. 6. In regard to *sensitiveness.* When recent a slight flexion may induce very great tenderness. Usually the degree of tenderness is proportionate to the degree of the flexion. 7. In regard to *duration.* In an old standing case sensitiveness may have been lost. In recent cases there is greater congestion. In recent cases also, atrophy at the seat of the bend has not become very evident, whereas in long-standing cases, the uterine wall may be very thin at the seat of the flexion. 8. Occasionally there is a slight *lateral* tendency of the retroflexed uterus. 9. In regard to *complications.* Perineal rupture is a serious complication; peritoneal adhesions binding the uterus down in its abnormal position are of great importance.

The *course* of retroflexion is chronic. The course is usually from bad to worse unless suitable treatment is adopted. Its effect on the patient is in many cases most disastrous. Incapacity for exertion, for the ordinary business of life, helpless invalidism, these are the effects witnessed in long-standing severe cases. The patient has often become an invalid before the nature of the disorder is known or even suspected. Its commencement is often insidious. "Spinal weakness," "hysteria," dyspepsia," under these or other names the disorder establishes itself, and not uncommonly has existed for two or three or indeed many years before it is actually detected.

Symptoms.—There are no special symptoms of retroflexion, but this distortion can frequently be distinguished from anteflexion by analysis of the symptoms present. Pain or difficulty in locomotion (dyskinesia) is the commonest, and may be observed in all degrees of severity, beginning with cases of slight pain on walking some distance, and ending with cases where the slightest movement induces extreme suffering. Here it should be stated also, that in a few cases the condition approaches to paraplegia, the power of walking being actually impaired. This *reflex paraplegia,* for such it seems to be, was mentioned as produced by this form of displacement some few years since by Dr. Priestley. I have observed the occurrence in a few cases. It is very important not to confound it with true spinal disease. And it must not be confounded with ordinary dyskinesia. In the latter case walking is possible, but produces pain; in the case of reflex partial paraplegia, the power of walking is lessened, and the action of certain muscles is imperfect.

The pain on motion observed in cases of retroflexion is evidently due to the temporary increase of the flexion produced by the motion. It can always be predicated that certain exertions or positions of the body will produce pain, and the result is in conformity with this idea. Pain on defæcation, pain on sitting, on stooping, &c., all belong to this category.

Obstruction to micturition is not uncommon. Retention of urine may occur from time to time, but it is not a constant symptom.

Pain in the sacral region is common, pain in the hypogastric lateral regions is sometimes also noticed in a very acute degree. Pain extending down the course of the sciatic nerves on one side or the other is not uncommon.

Sickness or nausea is common. The severity of this symptom varies much. The patient is generally most sick when the flexion is greatest. It may threaten her with dissolution from its long continuance. It is not a constant symptom.

Liability to abortions.—Retroflexion frequently prevents conception, but conception having occurred, abortion very frequently follows unless the case be understood and properly treated. Abortion is more certain to occur in retroflexion than in anteflexion cases, that is to say when left to themselves.

Sterility is a common result.

Menstrual derangements.—Menstruation is often profuse; clots are often observed; in some cases a profuse flooding occasionally happens. Often the duration of the discharge is prolonged, sometimes even for two or three weeks. Dysmenorrhœa is not seldom met with, and may be very intense. In some cases the discharge is too scanty and of bad colour. In a few cases amenorrhœa is produced, menstruation actually ceasing.

Leucorrhœa of a very troublesome character is often observed. That variety which may be described as "puriform leucorrhœa occurring in gushes," is rather common in chronic cases of retroflexion, the discharge consisting of retained puriform secretions from the interior of the uterus. In other cases it is the result of the congested condition of the cervix and of the glands there situated.

Difficulty in defæcation, sometimes amounting to obstinate constipation, is common. Painful defæcation is rather common, every straining effort increasing the flexion. In some cases a constant tenesmus or diarrhœa results.

Pain on intercourse (dyspareunia) is common. Hysterical phenomena of great intensity are occasionally observed. In a few cases convulsions occur. These reflex phenomena appear to depend on the compression of the tissues of the uterus at the seat of the flexion.

Diagnosis.—In cases where the flexion is in the first degree, and the uterus not much lowered in the pelvis, the diagnosis is difficult, unless the sound be used. But ordinarily there is no difficulty, the exploring finger readily defines the outline of the fundus in its abnormal position. A vaginal or rectal examination or both should be made. The condition principally liable to be mistaken for retroflexion is fibroid outgrowth low down at the back of the uterus. Other tumours —pelvic hæmatocœle, pelvic cellulitis, uterine fibroids pendulous behind uterus, small ovarian tumours, prolapsed ovary, &c., do not resemble the fundus uteri in outline or consistence. The sound conclusively proves the nature of the malady, but is not available when pregnancy is possible.

Prognosis and Treatment.—The curability of a long-standing case of retroflexion is always a matter of doubt. When the organ has been seriously distorted for some years, and the thickness of the walls at the point of flexion materially reduced, particularly so. Whereas a case of a few months' standing only is usually readily curable. The well-known plasticity of the organ is a great help in the curative process.

General treatment is usually imperatively necessary, whether local treatment be employed or not. Tonics, and a careful restorative dietary necessary for general weakness and mal-nutrition, are usually required in these cases.

The positional treatment is to be conducted as follows:—As a rule the patient should maintain the horizontal position, lying on the side, and frequently partly on the chest, in the prone position on an incline of pillows. Sitting is injurious. Very short walks may be permitted in not very severe cases. Stooping, lifting, or other such exertions are prohibited.

Mechanical internal treatment is necessary in cases where the malady is established, and it may be conducted in several ways.

Occasional replacement of the organ and unbending it by the sound, together with positional treatment, answers well in some cases. The sound should be very gently used, for mischief may otherwise be done.

But in most cases a pessary is required as an aid, and in very many is essential. The best pessary is the Hodge-shaped pessary, of size and form adapted to the case, by which the fundus is pushed upwards, and aided by positional treatment it is capable of effecting frequently almost all that is desired. It should be worn continuously. The sound may be used in conjunction with the wearing of a pessary. In a case where the uterus has become hardened the Hodge pessary alone and unaided by positional treatment and use of the sound will fail. Copper-wire rings $\frac{1}{12}$ inch, covered by india-rubber, so as to make the whole thickness $\frac{5}{12}$ of an inch, and made of various sizes, answer admirably, as they can be bent into any required shape. Other materials are often used. As regards vaginal pessaries, it is necessary to bear in mind that the fundus may be bent backwards over the top of the pessary unless the pessary be sufficiently long. The sound offers the only means of ascertaining if the flexion be really controlled by the pessary. If the flexed uterus be hard the pessary simply pushes up the fundus without unbending the uterus.

Intra-uterine stem pessaries are sometimes employed, but require great care. They are not so suitable for retroflexion as for anteflexion.

Dilatation of the uterus by tents is sometimes employed as preliminary to other treatment.

The treatment is often required to be spread over a long time in order to completely restore the uterus to health. The malady is one prone to recur. Pregnancy occurring in the course of the case is favourable, but the flexion will probably re-appear after the pregnancy is over and must be kept in check by suitable treatment.

The congestion attendant on retroflexion is treated by some with leeches or scarifications. But this congestion disappears when the uterus is straightened. The congestion is mechanical in its origin and quickly subsides on placing the patient in a favourable position, or on elevating the fundus posteriorly by means of a pessary.

The hypertrophy of the os uteri and of the uterine body often present in long-standing cases is very difficult to get rid of, but it usually gradually lessens as the shape of the uterus is improved.

LATERIFLEXION OF THE UTERUS.—Lateral displacement and flexion of the uterus generally occur to only a very slight extent. It frequently happens that in anteflexion the fundus is distinctly inclined a little to one side; and the same holds good to retroflexion. But distinct lateral flexion is very rare. Tumours may of course displace the uterus in the lateral direction.

The slight lateral direction which the fundus sometimes takes whether in posterior or anterior flexion, is however a practical matter of great importance, for it is always more difficult to treat such cases successfully than those in which the flexion is directly backwards or forwards. Modifications in the shape of the pessary employed are requisite, and advantage must be taken of the influence of gravity by placing the patient in a proper position to obtain the benefit of it. Thus in a case of retroflexion tending to the right side the prone position inclining to the left would materially help in the management of the case.

CHRONIC INVERSION OF THE UTERUS.—Chronic inversion of the uterus is generally the result of labour, the organ becoming inverted at or after the removal of the placenta.

The inversion may be complete or partial. Its acute stage cannot be described in this place. In its chronic form it is liable to be met with months or years after a particular labour.

VARIETIES.—The organ is completely inverted, so that no portion of the uterine cavity remains as such, or it is a partial inversion.

COURSE, SYMPTOMS, AND DIAGNOSIS.—The malady has no tendency to a spontaneous cure, the uterus becomes hardened and firm in its distorted shape, atrophy of the compressed portions of the uterine wall and hypertrophy of others result. In complete inversion there is less alteration of this kind. The symptoms consist of pain and "bearing-down" more or less continuous, but not present to a severe degree after a few months in all cases; almost continuous losses of blood, sometimes very profuse, alternating with puriform discharges; general discomfort and dyskinesia; menstrual periods not so distinct as usual from hæmorrhages. These symptoms persisting in greater or less severity, and dating from a particular labour. The symptoms are very similar to those of uterine polypus.

The diagnosis can be established by examination only. A rounded tumour presents at the os uteri (partial inversion), or fills the vagina, and appears to grow from the upper extremity of the vagina (complete inversion). In complete inversion the sound cannot possibly be passed beyond the pedicle. In partial inversion the sound enters half an inch, or an inch perhaps, beyond the constricting ring. This latter circumstance distinguishes it from polypus; for in cases of polypus the sound passes the full distance into the uterus. To this statement there is one exception, viz., in the case of a polypus which is complicated with partial inversion. Double examination per rectum and suprapubic, or by a sound placed in the bladder, shows that the body of the uterus is wanting from its proper position, and a depression can usually be felt superiorly, marking the spot of inversion. In doubtful cases this double examination is of great value.

TREATMENT.—Formerly the only treatment followed was removal of the tumour by excision, or the whip-cord ligature. Of late years very many cases have been successfully and completely cured by skilfully-applied continuous pressure, whereby the inversion has been removed. Difficulties in the way of this treatment are greater in some cases than others. The inverted uterus has been restored after fifteen years' duration of the malady, and in very many cases after shorter intervals. The functions of the organ have generally also been fully restored by the reduction. The pressure does not succeed in some cases. To aid in the process it has been recommended to incise the uterus bi-laterally at the situation of the os (Marion Sims and Barnes). When reduction cannot be effected, the whip-cord ligature may be applied. Dr. M'Clintock's plan is to place a strong ligature round the pedicle for three days, and then to excise the tumour.

HYPERTROPHIC ELONGATION OF THE UTERUS.—(*See* PROLAPSUS.)

PROLAPSUS OF THE UTERUS.—Prolapsus of the uterus is a general term, implying descent of the organ within the pelvis, or a further descent—actual protrusion externally. By some writers the term "procidentia" is applied to the latter class of cases.

There are various forms of the affection. It is rarely simple, for there are very few cases in which the uterus, unchanged in form or size, descends to any considerable extent: the majority of cases are complicated.

SLIGHT INTERNAL DESCENT OF THE UTERUS. —Prolapsus in this form is an almost invariable accompaniment of flexion of the uterus, whether forwards or backwards. The organ as a whole is too low in the pelvis. Another associated condition is enlargement of the uterus, *e.g.*, retarded involution after parturition, or simple hypertrophy, the result of long-standing chronic congestion. Another possible associated condition is rupture of the perineum, produced by some former labour. It is rare to meet with the uterus low down in the pelvis, except in association with one of the conditions here mentioned.

CONSIDERABLE DESCENT OF THE UTERUS.—Here the organ is so low that it is on the point of projecting at the vulva. This is a condition very rarely met with, except in association with decided flexion, chronic enlargement of the uterus, or considerable deficiency of the perineum.

CYSTOCELE.—In cases of cystocele the floor of the bladder is protruded at the vulva in form of a round soft tumour. In these cases the uterus is always, according to the writer's experience, in a state of very decided ante-version or flexion, and in most of the cases there has been rupture of the perineum. But acute anteflexion may produce it when the perineum is sound. The uterus is quite low down, and close behind the bladder.

RECTOCELE.—Here the tumour at the vulva is a portion of the rectum. It is usually produced by the combination of retroflexion of the uterus and rupture of the perineum. The uterus is very low down in its retroflexed state, and close to the perineum.

COMPLETE PROLAPSUS (PROCIDENTIA.)—Here the uterus is outside the vulva. It is usually the case that the uterus is protruded in the retroflexed state. To the majority of such cases the following description applies:—The enlarged retroflexed uterus, which has for some time occupied a very low position in the pelvis, finally escapes altogether from it, especially if the natural barrier to such escape—the perineum—be deficient. One proof of this is that, in reducing the prolapsus, the uterus is found acutely retroflexed. Anteflexion of the uterus is not found in complete prolapsus.

The uterus is often of great size in these "complete" cases. The mucous membrane covering it is often much ulcerated, and abraded from external friction.

PROLAPSUS WITH HYPERTROPHIC CERVICAL ELONGATION.—This condition, first well described by Huguier, presents two or three varieties. The *vaginal portion* of the cervix uteri, alone, may be much too long and protrude externally. It may occur in single or married women, or the *supra-vaginal* portion of the cervix may be so elongated and hypertrophied, as well as prolapsed, that a large truncated tumour extends out of the vulva, and the whole length of the uterine cavity from external os to top of fundus may be five to seven inches. The greater part of the canal so elongated is the cervix. Properly speaking, these are cases of descent, not necessarily of the uterus proper, but of elongation and descent of the cervix. Laundresses and cooks are particularly subjects of this variety of prolapsus.

ETIOLOGY.—Prolapsus of the uterus is the result of the operation of causes generally complex. The following are directly or indirectly causes:—

1. *Pressure downwards.*—This may be made so excessive as to cause prolapsus in many ways. *a.* Extra weight of the uterus, *b.* extra weight of parts above (*e.g.* intestines, omental fat, ascites), *c.* unusual straining force applied above as in straining efforts in defæcation or micturition, straining induced by flexion of the uterus (often very great), *d.* excessive strains on abdomen in lifting weights, *e.* violent propulsion of uterus downwards in cases of accident—*e.g.* falling on the nates.

2. *Defective resistance.*—Under this head come *a.* loosening or relaxation of uterine ligaments, *b.* destruction partial or complete of vaginal floor, viz., perineal rupture, *c.* absorption of fat near vulvar aperture, *d.* general debility impairing muscular resistance at vulvar aperture.

3. *Operation of a dragging-down force.* 1. Presence of a prolapsus. 2. Hypertrophic elongation of the cervix. 3. Tumours growing at the os uteri.

TREATMENT.—The treatment of slight internal prolapsus consists generally in treatment of the anterior or posterior version or flexion which is so generally present. This treatment is however rendered difficult in many cases from the presence of perineal rupture, so that a vaginal pessary is not so readily or satisfactorily applied. Hence larger pessaries are requisite when the perineum is defective. In cases of extensive rupture of the perineum a plastic operation to restore the lost support is necessary.

In the cases of complete prolapsus it is essential to the success of the treatment that the shape of the uterus be attentively considered. For instance, if there be a chronic severe flexion of the uterus it will be of little service to restore the perineum by operation unless the flexion be at the same time or subsequently treated; otherwise the protrusion will be certain to recur.

When perineal operations are required, as they frequently are, the operation must be carefully adapted to the end in view. It is frequently necessary at the same time to reduce the size of the vagina by taking up a portion of the floor.[1] Thus the size of the vulvar aperture and of the vagina are at one and the same time reduced.

Many cases of severe prolapsus are susceptible of relief by application of carefully adjusted pessaries or external supports of various kinds. For severe cases of prolapsus not admitting for various reasons of absolute curative treatment Zwank's pessary is very serviceable.

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[1] Author's work on Diseases of Women, 3rd edit. p. 293.

DISORDERS OF UTERINE FUNCTIONS.

By Graily Hewitt, M.D., F.R.C.P.

Amenorrhœa.

Menstruation is entirely absent when the ovaries are wanting or when the uterus is imperfectly developed, or when, the os uteri or the vagina being closed, there is no outlet for the menstrual fluid. In such cases the amenorrhœa is complete. But the menstrual discharge having been established may become much diminished, or very scanty and irregular, or it may prematurely cease; and this cessation may occur at any period. The term amenorrhœa is also applied to the latter classes of cases.

Diagnosis of the Cause of Amenorrhœa in cases where no Menstruation has ever occurred.—Ordinarily puberty arrives and menstruation occurs before the age of sixteen or seventeen. But in some instances it is delayed, and the primary appearance occurs a few years later. These are cases of delayed puberty, and at first it is not possible without internal examination to pronounce an opinion upon them. Another class of cases are those in which the menstrual secretion is pent up and cannot escape. These cases are characterised by presence of pains in the uterine region, generally occurring particularly at monthly intervals, and the pains may be very intense. The diagnosis of the latter class of cases could not be arrived at without an internal examination. The degree of development of the uterus is ascertained best by a double examination—one finger placed in the rectum, and the catheter in the bladder. Retention of catamenia usually involves the presence of a swelling in the hypogastric region, due to the distension of the uterus. There may be also a distension of the vagina when the hymen is imperforate. It must not be forgotten that pregnancy is possible even in cases where no menstruation has yet occurred.

Imperfectly Established Menstruation.—This category includes very many cases. The discharge is imperfect; occurs perhaps once or twice in very small quantity, and not again; or there is an occasional scanty discharge at long intervals. In this class of cases the phenomenon termed *vicarious menstruation* is liable to occur, by which is meant that at the usual monthly period a sanguineous fluid escapes, usually from the stomach or from the lungs, or from some other internal mucous surface, or it may be from the surface of an external ulcer.

In cases of imperfectly-established menstruation the cause is *general* or *local*, or both combined. In many cases the amenorrhœa is the result of general feebleness, insufficient food, &c. Growth is rapid, and much nourishment is required. *Chlorosis*, as it is termed, is sometimes observed under these circumstances, the amenorrhœa being coupled with a very peculiar greenish yellow appearance of the skin. There is present in these chlorotic cases a profound anæmia, great feebleness, tendency to headache, coldness of extremities, &c. But amenorrhœa may occur in young women also, as the result of over-exertion, walking, riding, &c., in which cases it appears to be due to disturbance of the position and shape of the uterus. The soft, imperfectly-nourished uterus (see p. 666) readily gives way under excessive physical pressure. Thus, when the system is not well sustained, and there is too much exertion, one possible result is amenorrhœa.

Suppression.—One form of amenorrhœa is that arising suddenly from taking cold, standing on a wet floor, taking a cold bath during the menstrual flow, &c. Mental emotion is also a cause of suppression. Amenorrhœa arising in this way is not seldom serious in its effects, or difficult to cure. It seems probable that some internal change of an important kind must occur in these cases, the part affected being the uterus, or the ovaries, or both; or what seems not unlikely consisting in an inflammatory effusion of lymph over the peritoneum, covering the ovaries, and perhaps extending into the Fallopian tubes. Suppression may occur also from fevers; in a more gradual way it may occur from extreme weakness, however induced, from phthisis, &c.

Chronic flexion of the uterus is occasionally observed to produce complete cessation of menstruation, and lead to its premature termination, this result arising from the compression and hardening of the organ, and the great obstruction of the uterine canal.

Lastly, it must be stated that amenorrhœa may occur from change of air, or place, or habits of life, without further ascertainable cause; that it may result from fibrous tumour, or chronic hypertrophy of the uterus; from cystic, or other diseases of the ovary.

Treatment.—Amenorrhœa arising from absence or original defect of the generative organs is rarely susceptible of cure. In cases of infantile uterus Simpson's treatment by the use of galvanic intra-uterine stems has in some cases succeeded.

Amenorrhœa and retention, due to imperforate condition of the hymen or vagina, or cervix uteri, are cured by operation. In these cases the aperture should at first be made very small, so as to allow of a very gradual escape. It may afterwards be enlarged.

In amenorrhœa from suppression hot hip-baths should be used, and the patient kept in bed at the proper period. Mustard may be applied to the thighs, warmth to the hypogastrium. Leeches applied near the groins are sometimes indicated. Special emmenagogues may also be employed. Any discovered internal disorder should of course be appropriately treated.

In cases of amenorrhœa due to flexion of the uterus the distortion must be suitably treated. Softness of the uterus is likely to be associated with amenorrhœa, and slight flexion, particularly during the first two or three years of menstrual life. It will be found in such cases that complete rest in the horizontal position is extremely efficacious in inducing return of menstruation. The dorsal horizontal position is usually the best. Great care in regard to diet is required, for serious defect in nutrition is the most important element in such cases. As a rule cases of amenorrhœa in young women are best treated by rest, good feeding, and fresh air. Experience shows that long walks or violent exercise of any kind are detrimental.

The best emmenagogues are tonics, such as iron, quinine, good food, and careful general hygiene. Iron in various forms, but in small doses, is usually well borne. Careful attention to the bowels is always required, and occasional mild aperients may be necessary. In cases where the liver has become disordered, a mild mercurial may be given with advantage at long intervals. Special emmenagogues should only be given when other more simple remedial measures have failed. Aloes, myrrh, essential oil of savin, ergot, are the emmenagogues most generally in repute. Mustard cataplasms on the thighs, hot hip-baths, with or without mustard, injections of aloes *per rectum*, injections of hot water *per vaginam*, are valuable adjuvants.

DYSMENORRHŒA.

Dysmenorrhœa implies presence of pain or discomfort in the pelvic region, at or about the time of the menstrual discharge. The menstrual discharge is also almost invariably abnormal in some other particular, *e.g.*, it is scanty, or almost entirely absent, or it is delayed beyond the proper time, or it is profuse, or it is interrupted. The pain varies in intensity, sometimes amounting to slight discomfort only: in cases of an extreme character, being so severe that the patient is beside herself with the indescribable agony experienced. And between these two extremes all imaginable gradations are observed.

The seat of the pain is not constant. The sacral region is generally the part spoken of, but in severe cases the uterine region is referred to. The pain is as a rule intermittent in character.

The cause of the pain has been the subject of much dispute, but of late years the balance of opinion has been strongly in favour of the view that dysmenorrhœa is the result of an obstacle to the escape of the menstrual fluid. The pain is due to the attempts made by the uterus to expel its contents, the uterus contracting, and being thereby thrown into a state of spasm ("uterine colic" of the older writers).

The pain is relieved when the uterus succeeds in expelling the whole or a part of its contents. The obstruction to the escape of the fluid is produced by different causes, but the most common cause is flexion of the uterus, producing compression and constriction of the uterine canal at or near the internal os uteri.

The uterine canal at the internal os is naturally small; the canal is surrounded by dense unyielding tissues; bending of the uterus implies narrowing of the canal at this situation, which narrowing becomes so considerable in many instances, as to amount to virtual closure; and the menstrual fluid and *débris* find no sufficient outlet. Further circumstances often lead to temporary exaggerations of the flexion at the menstrual period, acute congestion of the body of the uterus may set in, and the whole organ becomes swollen. Hence the menstrual evacuation is more and more hindered. Again, a quantity of menstrual fluid having collected *in utero*, the body of the uterus becomes unnaturally distended, and hypertrophy with dilatation follow. Clots not seldom form in the uterus as a result of the delayed evacuation, and their exit is attended with terrible suffering. Dysmenorrhœa may lead eventually to complete suppression of menstruation, but in some cases it leads to the precisely opposite effect, viz., very profuse losses (menorrhagia), the result of dilatation and hypertrophy of the uterus.

Flexion is the common cause of obstructive narrowing of the internal os. But there may be congenital narrowing at this situation, and small fibroid tumours sometimes grow in this locality and induce severe dysmenorrhœa.

The external os uteri may be the seat of obstruction. Congenital narrowness of the external os occurs in a few cases, with dysmenorrhœa as a result.

It appears that in some few cases the pain during menstruation depends on some morbid condition of the ovaries. Chronic inflammation of the pelvic peritoneum may produce exudation over, and thickening of the covering of the ovary—ovulation is thus impeded and pain would result. Probably in some cases also pain during menstruation arises from actual passage of blood from the uterus into the peritoneal cavity, giving rise to pelvic hæmatocele of the intraperitoneal variety.

It follows from what has been stated, that the large majority of cases of dysmenorrhœa are really cases of *menstrual retention*. The clinical evidence in favour of this conclusion is overwhelming, for the pain always undergoes mitigation or complete arrest when procedures are taken, or measures adopted calculated to provide a more easy passage for the menstrual fluid from the uterus.

Concurrently with dysmenorrhœa, serious *vomiting* is not rarely observed. Nausea is a common symptom.

Sterility is commonly conjoined with dysmenorrhœa, impregnation does not occur, because the patency of the uterine canal is interfered with by the flexion usually present. In some cases the sterility has probably its explanation in the fact that the uteru s constantly contains retained morbid products.

Membranous Dysmenorrhœa is a term applied distinct membranous cast of the uterine cavity is be in one piece or in several pieces. In these lining of the uterus appears to have a thicker str and when thrown off, does not, as under ordina break down into impalpable shreds. The expulsion of this thi lining gives rise to severe pain.

TREATMENT.—In slight cases general measures may suffice. Of these the most important is rest in the horizontal position; on the back if it be a case of anteflexion, on the side or on the face if the uterus be inclined backwards. If the state of the uterus be not known, the horizontal position on the side may be safely recommended. Care should be taken to keep the bowels unloaded. Opiate injections per rectum or morphia suppositories are very useful in relieving the pain. Ether, lavender, henbane, cannabis indica, are remedies which may be used. Warm fomentations, vaginal injections of rather hot water (100° Fah.), or the hot hip-bath are useful adjuncts. These latter procedures are also serviceable in the more severe cases. A popular remedy is gin and water, the efficacy of which, it may be conjectured, depends on its increasing the quantity of urine, thereby distending the bladder and helping to straighten the uterus; such an explanation at least would hold good in cases of anteflexion of the uterus. Guaiacum, black hellebore, colchicum, camphor, are other remedies strongly advocated by various practitioners of repute.

If the case be one of marked character, and the uterus is certainly flexed, the case must be dealt with appropriately. In quite young women, 16—18, general treatment may suffice, the uterus is probably very soft and the nutrition low. The case may, in some cases, be treated without special reference to the dysmenorrhœa, rest, good feeding, and a general restorative system of treatment being enforced, while the dysmenorrhœa, treated only by palliative measures, in time ceases. The very best effects result from such treatment, but only in these cases.

On the other hand where the patient is older, or where the uterus has become hardened in its distorted shape, general measures are useless from a curative point of view. The uterine canal must be mechanically straightened, and thus dilated in order to relieve the dysmenorrhœa. In some few cases bi-lateral incision of the internal os is practised, but this procedure is only useful when followed by the use of a stem pessary. As a rule the combination of occasional dilatation by a metallic dilator, with use of a suitable vaginal pessary, affords satisfactory results.

Cases of congenital or acquired narrowing of the external os uteri requires operative enlargement of the aperture by the knife or by scissors.

MENORRHAGIA.

The term menorrhagia implies excessive loss at the menstrual periods. The average quantity may be only slightly exceeded, or it may amount to a profuse flooding. The loss may be profuse and be limited to the ordinary number of days, or the period may be both profuse and very protracted, extending to a week, or even a fortnight. In many cases clots are expelled together with blood. As a rule the discharge is somewhat intermittent, there being alternate gushes of discharge, followed by periods during which the discharge is little.

The effect of the repeated losses in severe cases is to produce a profound anæmia, general feebleness, impairment of function of various organs. The watery condition of the blood, usually present in long standing cases, is a cause of persistence of the menorrhagia.

ETIOLOGY.—Menorrhagia is sometimes due to *general* causes. Chronic visceral disease, *e.g.*, of the liver, of the heart, of the kidneys (especially Bright's disease) may produce it. Any general prostrating influence, *e.g.*, over-lactation, mental worry, fever, sexual excesses may give rise to it. Lead-poisoning, malaria, residence in tropical climates, are other general causes. Severe shock, mental or otherwise, may give rise to menorrhagia. But ordinarily menorrhagia is found to be dependent on local causes. The most common cause of menorrhagia is flexion of the uterus. In severe cases it is not uncommon to find the uterus much hypertrophied, its cavity enlarged, and its shape decidedly distorted. Menorrhagia is not seldom one of the most severe effects of anteflexion or retroflexion. The characteristic of such cases, is that the loss occurs in considerable intermittent gushes, often accompanied by clots. The uterus becomes distended with blood, the escape of which is prevented by the flexion. The organ then increases in size, and at the end of a certain time, having by its mere distension become somewhat straightened, the retained fluid escapes in a gush. The process repeats itself. In the end the uterine cavity becomes greatly enlarged, and in time the enlargement becomes permanent, the walls become thickened, and hypertrophy ensues in consequence of the greater muscular activity of the uterus (Dr. John Williams). The loss of the blood at each menstrual period may amount to a flooding. The loss of blood is perhaps liable to be greatest in cases of severe retroflexion, but in many cases of anteflexion it is very great.

Cases of severe menorrhagia are not seldom observed when repeated abortions have occurred in consequence of a chronic ante- or retroflexion; the circumstances then present favour the production of a chronic hypertrophy and distension of the uterine cavity, and menorrhagia of a very intractable character ensues. Defective involutions of the uterus, whether after abortion or after labour at full term, is an important causal element.

Chronic inversion of the uterus is another local cause of menorrhagia. Cancer of the uterus is frequently also attended with menorrhagia, but in these cases there is more generally also hæmorrhage at other than the menstrual periods also. Polypi and fibroid tumours of the uterus also may give rise to severe losses at the menstrual periods, but in such cases also hæmorrhage is liable to occur between these periods. The same remark applies to other diseases of the uterus.

It must be remarked that menorrhagia is sometimes associated with effusion of blood (hæmatocele) in the neighbourhood of the uterus. In some of these cases the blood appears actually to pass from the uterine cavity into the peritoneum. Distension of the uterine cavity, due to obstruction at the internal os (from flexion) may probably be set down as an occasional cause of this latter occurrence.

A fungous condition of the mucous membrane of the uterus is held by some authorities of repute to be a not uncommon cause of menorrhagia.

TREATMENT.—In cases where the profuse loss is dependent on general disease and not on local disorder of the uterus, attention must be directed to removal of the cause, and means taken to prevent, as far as possible, undue loss of blood. A general, tonic invigorating system, including careful feeding and avoidance of exertion, is required in cases of great debility. Iron is very necessary. Tonics and purgatives are frequently both required in cases of sluggish circulation and slow hepatic action. Change of air, hip-baths of tepid water, sponging the surface and moderate exercise are generally indicated. Above all rest in the horizontal position during the catamenial periods is desirable. Excitement of all kinds should be avoided.

During the period of the flow certain measures may be adopted to diminish the quantity of the discharge. Sulphate of magnesia, combined with dilute sulphuric acid, is a favourite remedy. Rather strong doses of tincture of perchloride of iron are often serviceable. Ipecacuanha, gallic acid, cannabis indica, and opium are other remedies recommended. A vaginal douche of hot water will sometimes prove useful. Ergot is proper in cases of relaxed atonic uterus; ice applied to the spinal column is a remedy also to be recommended in such cases. When the loss is so great as to amount to a flooding a vaginal plug may be used. Two or three yards of wetted bandage forms an efficient plug, to be inserted through a speculum. Iced water may be applied over the pubes in such cases. Ether, ammonia, and brandy may be given internally.

In cases where the menorrhagia depends on local uterine disease appropriate treatment must be employed. In cases of flexion of the uterus the remedy is to cure the flexion by straightening and opening up the uterine canal, whereupon the fluid readily escapes. Experience shows that the menorrhagia, though just at first a little increased by placing the uterus in its proper position and opening the canal, is speedily relieved. But in cases where the uterine cavity has become permanently distended, and its walls hypertrophied, the menorrhagia is not always cured so quickly. Other local disorders causing menorrhagia, *e.g.*, polypus, cancer, require special treatment. In certain obstinate cases of menorrhagia intra-uterine medication is sometimes practised. Styptics, as perchloride of iron, tincture of iodine, or actual caustics, as nitrate of silver or nitric acid (Atthill) have been employed. Preparatory to such procedure the cervix uteri is dilated, a precaution necessary to avoid possible passage of a caustic fluid into the Fallopian tubes (Savage and Routh).

LEUCORRHŒA.

The term leucorrhœa implies presence of a non-sanguineous discharge from the female generative passages.

Varieties. A.—In regard to SOURCE. In some cases the discharge proceeds from the *vulva* and parts surrounding the vaginal entrance. In others it proceeds from the lining of the *vaginal canal*. In others again its source is the os uteri and the lining of the cervix (*cervical*), and lastly it may proceed from the uterine cavity itself (*intra-uterine*).

B.—In regard to PHYSICAL CHARACTERS. Thus in some cases the discharge is a *watery*, or nearly or quite transparent fluid. In other cases it has a *puriform* character, presenting a more or less thick creamy consistence. Another important variety is the *offensive* discharge, which may be puriform or more or less watery or slightly tinged with red. Lastly must be mentioned the *intermittent* variety coming away from time to time in gushes, generally of a puriform character, sometimes offensive, sometimes more purely watery in consistence, and proceeding usually from the interior of the uterus. As a general rule the discharge in cases of leucorrhœa is a mixture of secretions from various parts of the generative passages. It may contain mucus in a tenacious condition and in compact masses formed in the cervical canal; epithelial *débris* also in greater or less quantity from the cervix or from the vagina.

ETIOLOGY.—Leucorrhœa is constituted as a rule by an increase in the normal secretion of the affected parts. The various portions of the internal generative tract produce secretions which differ in quality. Hence the variety in the physical characters of the fluid. The general causes may be first considered. General weakness or debility may produce leucorrhœa, the secretions being increased in quantity owing to the congested condition of the mucous membrane of the uterus or vagina or vulva. This general weakness may be associated with disease of large and important viscera, *e.g.*, the heart, lungs, liver, &c. It may be due to over-lactation, to semi-starvation, to that lowering of the general nutrition of the body which constitutes the first stage of phthisis. It may be present at a later stage of this latter affection. Residence in marshy or damp localities has often been noticed as favouring the occurrence of leucorrhœa.

The *local* causes are very numerous. In the first place must be mentioned *gonorrhœa*, in which case the discharge proceeds, at first at all events generally from the urethra and vulva, and is of a greenish yellow colour. Later on it may produce inflammation of the vagina or cervix uteri, or parts beyond, and give rise to a discharge having a somewhat different physical appearance. *Syphilis* may occasion leucorrhœa, vulvar or vaginal, and probably also it may give rise to intra-uterine irritation and leucorrhœa. Ulcerated surfaces are usually observed at the vulva or in the neighbourhood under such circumstances.

Flexions of the Uterus constitute a very common cause of leucorrhœa. Under these circumstances the leucorrhœa is cervical or intra-uterine, sometimes both. The cervix of the uterus, congested and turgid with blood, pours out from its interior a mucous alkaline secretion, often in great quantity, which, mixing below with the vaginal acid secretion, assumes a creamy appearance. This congestion of the cervix may be due to other causes than flexion, but the latter is its most common antecedent. Further, when the cervix has become thickened and hypertrophied the lining membrane is permanently in a state of engorgement, and increased secretion is the result.

There is a peculiar and very important variety of leucorrhœa, designated above as *intermittent*, which is usually connected with presence of flexion of the uterus, and deserves here a special mention. The secretions of the body of the uterus being prevented escaping readily owing to the flexion, a difficulty which is greater or less according to circumstances, accumulate *in utero*. Being retained they irritate the uterus, more fluid is secreted, and the cavity is more dilated. After a time, or in consequence of the position of the uterus becoming altered for the moment, the discharge escapes in a gush. This process repeats itself perhaps two or three times a day, perhaps only once in two or three days. It is more apt to occur during the week or two after menstruation. This intermittent leucorrhœa is an almost certain sign of the existence of an uterine flexion. Not unfrequently a pain is felt indicating uterine contraction concurrently with the escape of the fluid. Patients sometimes describe the occurrence as the breaking of an internal abscess. Further, it is the fact in some instances, that the discharge in these cases is very offensive, owing to decomposition of the fluid in the uterus. Such intra-uterine accumulations may probably occur when no flexion of the uterus is present, owing to agglutination of the os uteri, or of some part of the cervical canal. A continuous puriform leucorrhœa is frequently also produced by flexions directly or indirectly, congestion, turgescence, and hyper-secretion of the cervical and other glands being thereby produced. The majority of cases of obstinate leucorrhœa belong to this category.

Defective involution of the uterus is a not uncommon cause of leucorrhœa; which may be cervical or intra-uterine in origin. *Polypi of the uterus*, fibrous and mucous, may occasion leucorrhœa. Polypi give rise generally to a rather watery leucorrhœa, but there are many exceptions to this rule. *Ascarides in the rectum* may occasion leucorrhœa particularly in children, in which latter case there is vulvitis usually present. In children also leucorrhœa is liable to occur from simple debility, and its presence has occasionally given rise to unfounded suspicions of the disorder being a communicated gonorrhœa. *Hæmorrhoids, presence of fæcal accumulations,* &c., *vascular tumours of the meatus urinarius,* must be also included in the list of possible causes. *Cancer of the uterus,* epitheliomatous disease of the cervix, cancer of the vagina, &c., give rise to leucorrhœa, frequently offensive in character, sometimes (cauliflower excresence of the os) of a brownish watery character, frequently more or less red in colour.

Chronic inversion of the uterus occasions copious leucorrhœal discharges. *Congestion* of the uterus, as a whole, from whatever cause, *hypertrophy of the cervix, prolapsus of the uterus* in its various forms, are other causes of troublesome leucorrhœa. Excess in sexual intercourse and self-abuse are other possible causes of leucorrhœa.

Treatment.—The primary object in cases of leucorrhœa should be to remove the cause. This ought always to be sought for, and if possible removed.

General treatment is applicable in many cases, and if there be no special local irritating condition in existence, such general treatment alone may prove successful. Careful hygienic treatment, fresh air, moderate but regular exercise, sufficient nourishment, baths, frictions of the skin, change of air, regulation of the digestive functions and of the action of the bowels, are of great service. Habits of an injurious tendency must be corrected.

Local Treatment.—Injections are of great utility in all cases. The water employed is best used a little warm; a pint may be injected twice a day. Medicated injections containing alum, zinc, tannin, infusion of tea, nitrate of silver, are frequently employed. Medicated pessaries are also advantageously used in some cases. Most of the agents used are astringent in character; their action is usually limited to the vagina and os uteri; antiseptic injections are frequently employed when the discharge is offensive; tincture of iodine (one in ten of water), diluted carbolic acid, Condy's fluid, &c., are used under such circumstances. The hip-bath is an useful adjunct. Counter-irritation, in the shape of blisters or croton oil liniment to the sacral region, is sometimes of service.

If the leucorrhœa be found to be dependent on some local irritating condition the foregoing treatment will probably prove only palliative. An appropriate and more radical treatment will then be required.

SECONDARY EFFECTS OF UTERINE DISEASE.

INTERFERENCE WITH LOCOMOTION (UTERINE DYSKINESIA).—Impairment of power of locomotion is a symptom and effect of uterine disease so common and so important that it deserves to be considered separately and distinctly.

By uterine dyskinesia is meant an inability to walk or move, or perform certain of the ordinary motions of the body, without producing pain referable to the uterus, this pain being either situated in the sacral region or in the groin, and sometimes extending to the umbilicus. This inability varies exceedingly in degree, the pain or inconvenience produced by movement is sometimes slight, while in many instances it is very great, and in extreme cases it is so great that after a time the attempt to move is given up, and the patient becomes completely helpless.

Writing on the subject some years ago, the author described this condition under the name of "uterine lameness," but the appellation "uterine dyskinesia" is more comprehensive, and perhaps more accurate.

This inability to perform certain motions without feeling pain referable to the uterus is not a paralysis in any sense of the word, at least in ordinary cases; but it does appear that in some few cases, of which Dr. Priestley has related instances, and which the author also has witnessed, a paralysis may occur to a more or less complete extent. Generally, however, the patient is perfectly able to move or walk, but the exertion always gives rise to pain.

This symptom or effect of the presence of uterine disease is so common that it has been undeservedly passed over; and it has certainly in many cases been misinterpreted, patients complaining of it having been often thought fanciful when they were really describing very positive and tangible phenomena occurring in their daily and personal experience.

Uterine dyskinesia is observed in almost all cases of flexion or distortion of the uterus; and it appears that this condition of the uterus is its most common cause. The act of walking or stooping, or going up and down stairs, or any movement of the abdominal muscles, gives rise to a temporary exaggeration of the existing flexion in these cases, and thereby occasions pain in the back or groins, or down the back of the thighs, or in the region of the ovaries. Chaissaignac, writing on the subject of the cause of pain on motion, observed in cases of uterine "deviation," thought it due to the concussion or shakings of the uterus thereby produced. But the above explanation appears more completely to cover the phenomena observed in these cases. It is a matter of interest to observe that in those cases where the uterine dyskinesia is greatest, there is universally found to be the greatest amount of tenderness of the uterus—a tenderness recognisable on making a digital examination. In acute flexions the tenderness is often extreme, and it is precisely these cases in which uterine dyskinesia is a most prominent and noticeable symptom.[1]

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[1] In those cases, also, where excessive internal tenderness is present, intercourse is necessarily attended with more or less pain. "Dyspareunia," the term suggested by Dr. Barnes to describe this symptom, appears to be more frequently, according to the author's experience, connected with the presence of flexion of the uterus than with any other abnormal condition of the sexual organs.

Pain in the various situations about the pelvis, in the back, in the groins, in the region of the ovaries, has been long associated with uterine disease. By many those pains felt in the ovarian region have been considered, but erroneously, as the author believes, as generally indicative of ovarian inflammation or irritation. It is a remarkable fact, however, that pains in these situations are very rarely experienced when the body is in a state of repose. (The pains felt at the menstrual period are here excluded from consideration.) It is chiefly when the patient moves, or attempts an exertion, that the pain occurs, and it is on inquiry found to be the case that the particular exertion which brings on the pain is the one which would be likely to exaggerate and intensify an existing distortion of the uterus.

It is hardly necessary to observe that all pains in or about the pelvis produced by motion are not due to uterine disease. Undoubtedly when the ovary is displaced, when it is tender or inflamed, pain may be produced; pelvic peritonitis also is a condition which would give rise to pain on movement. Doubtless, also, there are other like causes. But the statement that in the vast majority of cases these pains are connected with the particular disturbance of the uterus above described, will be found amply supported by clinical observation.

Further analysis renders it evident that there are two possible reasons for the circumstance that uterine flexions give rise to uterine dyskinesia. The existing flexion is always accompanied by more or less congestion, distension of the uterine tissues, and compression of certain other portions. On the whole, evidence is in favour of the conclusion that it is the increased compression of uterine tissue at the seat of the flexion which is the principal cause of the actual pain. When the uterus is acutely flexed and intensely congested the nerves are everywhere more or less affected; but at the seat of the flexion there is greater compression than elsewhere. Experience shows that the pain on motion does not occur, or is very perceptibly diminished, when this compression is removed; when, for instance, the uterus is made straight, and the condition causing such compression ceases to operate in this way.

Uterine dyskinesia is one of the earliest symptoms of uterine flexion, and as such it is a symptom which has an extreme importance.

REFLEX NERVOUS DISTURBANCES DUE TO UTERINE DISEASE.—There are two symptoms observed in certain cases of uterine disease which may be classed under the above head—*hysteria*, and *nausea* or *vomiting*. They both appear to be in reality nervous disturbances of a reflex character, the inciting agent being some abnormal condition of the uterus.

Hysteria.—The idea of the association of hysteria with disorder of the generative organs is of ancient origin, as the name itself implies. And although writers may be found who would contend that the generative organs are not concerned in the production of hysteria, this opinion is not one shared by those possessing most acquaintance with the subject of uterine and ovarian disease.[1]

Without professing to deal with exceptional and rare instances, those, for instance, of hysteria in the male sex, it will at once of course be conceded that hysteria is a disease *par excellence* of the female sex. Clinical observation shows that in the very large majority of cases hysteria, or hysterical manifestations, are observed in connection with some abnormal condition, or some irritation proceeding from the sexual organs. Assuming, therefore, that this view of the case is correct,—space renders it impossible to discuss this particular question at the necessary length—we may inquire what this abnormal condition or irritation actually is?

The author has formed an opinion on this subject which may be formulated as follows:—

In cases where the hysteria is more or less chronic, and more or less constantly present, where the patient is liable to paroxysmal attacks, hysterical in character, the exciting cause—the irritation, in fact—will be found to be a well-marked flexion of the uterus.

This opinion is based on clinical observation extending over upwards of seven years; during which time a tolerably large number of cases, such as those above alluded to, have been subjects of observation. The cases have one and all resolved themselves, on proper inquiry and investigation, in the direction above indicated; and the testimony of these cases has been so uniform and strikingly similar, that the author can have no doubt that the generalisation in question is a sound one.

More than this, it has been observed in these cases that the tendency to hysterical attacks or manifestations has uniformly disappeared on measures being taken to relieve the disorder present, viz. to cure the existing uterine flexion.

The cases comprehended in the above description were of what may be termed typical character, and characterised by a tendency to repeated attacks or paroxysms. Concerning other cases in which hyperæsthesia, anæsthesia, and various slighter symptoms of hysteria are observed, the author has nothing to give as the result of actual personal observation, not having encountered such cases under circumstances where an internal exploration was possible; and he has had no means of testing the truth of the generalisation above given in regard to its application to the slighter symptoms of so-called hysteria. Meanwhile the above statements are given for what they are worth; what that worth may be can be tested without any great difficulty by the observations of others.[2]

The hysterical paroxysm, according to the author's view, is the result of a reflex irritation. That irritation consists in the mechanical compression of certain portions of the uterine tissues. Such compression is produced in cases of uterine flexion by the alteration of shape which the uterus undergoes.

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[1] See, for the other side of this question, article "Hysteria," in vol. ii.—EDITOR.

[2] See also author's work on Diseases of Women, 3rd edition, p. 306.

It may be inquired, Why is it that the compression of uterine tissues produced by flexion does not invariably produce hysterical manifestations? It is to be replied that reflex movements are not in all cases and in all individuals produced by the same apparent conjunction of circumstances. Convulsions are not an invariable result of the presence of lumbrici in the intestinal canal, but no one disputes the fact that they do occasionally produce the reflex phenomena in question. Probably there is a predisposition or diathesis involved—a greater excitability in some cases than in others.

A noteworthy fact must further be stated. It will be observed in cases of liability to paroxysmal attacks, that physical exertion, such as would be likely to exaggerate the existing flexions, is in these cases almost always found to produce hysterical phenomena. There is, in fact, a constant experiment going on before the eyes of the observer, and the patient has only to be questioned as to her daily experience to extract facts which bear out the accuracy of the foregoing statements.

It is further to be remarked, that convulsive attacks, non-hysterical in character, are observed in certain rare cases to be produced by uterine flexion. Such convulsive attacks resemble hysterical attacks in the mode of their production—temporary exaggeration of the existing flexion being their exciting cause—and in point of fact it is difficult to draw a line between these and ordinary hysterical convulsive attacks. The attacks are in both cases the result of irritation in the uterus acting in a reflex way on the spinal system, and thus exciting convulsive movements.

The recent writings of Professor Charcot on the subject of hysteria have attracted much attention. Professor Charcot apparently considers the ovaries the seat of the morbid irritation. The author regrets that as yet he has had no opportunity of personally investigating the causes on which Professor Charcot founds his conclusions.

Nausea and Vomiting.—There are few cases in which the uterus is the seat of disease in which nausea or tendency to vomiting is not observed. The sympathies existing between the uterus and the stomach have been long known and recognised, but it is not yet so generally well known as it deserves to be that sickness or nausea are very commonly symptoms of uterine disorder.

This symptom is observed in all degrees of intensity. The nausea or sickness is sometimes very slight, only noticed perhaps on first moving in the morning; or it may be so severe that the patient is on the point of perishing from the long-continued deprivation of food. In other cases the slight but more or less constant nausea present has the effect of producing a slow but very insidious form of starvation.

The particular condition of the uterus, which most commonly gives rise to nausea or vomiting, is alteration of its shape. Marked flexions of the uterus are almost invariably attended with nausea or vomiting, and those cases in which it is most marked are the cases attended with most congestion of the uterus and most disturbance of the circulation within its tissues. The imperfectly nourished uterus, which is soft and pliable to an unusual degree, and which readily bends when the patient moves or exerts herself, frequently produces a chronic liability to nausea which is most characteristic. Nausea and vomiting may doubtless be produced by other morbid conditions of the uterus, for instance, in some cases of fibroid tumour, the symptom is observed. In cancer of the uterus, sickness is often a marked symptom. Again, certain affections of the ovary—notably marked displacement of the ovary—may occasion it, but these other operative causes are of not very frequent occurrence.

The clinical proofs of the connexion existing between uterine flexions and nausea and vomiting, as cause and effect, are, according to the author's experience, most convincing. The symptom in question is not invariably present in cases of uterine flexion, but it may be asserted that when marked sickness is present in association with uterine disease, the particular disease present will almost invariably be found to be acute flexion of the organ. It will be found further in such cases that every movement of the body calculated to intensify or aggravate the existing flexion, will assuredly increase the vomiting or nausea. The erect position is sufficient in many cases to produce it; hence the occurrence of nausea on first rising in the morning. While, on the other hand, the nausea or vomiting ceases, or undergoes marked mitigation under opposite circumstances—when, that is to say, the patient is made to rest in such a position that the flexion is relieved.

Severe sickness is occasionally observed at the menstrual period in conjunction with marked dysmenorrhœa. The vomiting is sometimes most intense and distressing. It appears to be due to the combined effects of the distension of the uterine cavity with retained blood and increased compression of the uterine tissues at the seat of the flexion. The diagnosis of uterine sickness from other varieties of the affection is generally a matter of no difficulty. One characteristic of uterine sickness is that it is brought on by some motion of the body, rising in the morning, stooping, sitting at table, standing or walking, and that it subsides or does not occur at all when the patient remains in the recumbent position. But in cases of retroflexion of the uterus causing sickness, the sickness goes on notwithstanding the fact that the patient is lying down. A distinction must be drawn, therefore, and it is remarkable how accurately in many cases a diagnosis may be made as to the nature of the malady from the effect of change of position on the sickness. Thus in cases of anteflexion the sickness does not occur while the patient is in the dorsal position. On the other hand, the sickness is abated almost immediately in a case of retroflexion of the uterus by placing the patient in the prone position.

The nausea or vomiting above described appears to be the result of reflex irritation starting from the uterus. The particular nature of the irritation thus potent in exciting the gastric symptom in question, is an interesting question. Assuming that flexion of the uterus is the cause, it remains to explain how and why it is that the flexion operates in this way. The author is decidedly of opinion that the compression of the uterine tissues, including the nervous fibres therein contained at the seat of the flexion, is its essential cause. That mechanical compression of such nervous fibres as exist in the tissues situated at the central part of the uterus around the internal os uteri, must ensue when acute flexion occurs is a matter which admits of no doubt. All clinical evidence, too, is in favour of the conclusion that this part of the uterus is often in a state of very undue sensitiveness in cases of flexion, as proved by the use of the sound. The subsidence of the sickness on straightening the uterus is also in favour of this view of the matter. Besides compression at the seat of the flexion, there is stretching and distension of uterine tissue (involving also nerves) at other situations, in consequence of the congestive and circulatory obstruction. It might be properly argued that reflex irritation would be likely to be set up by such tension of the uterine tissues generally, and it must be admitted that this is a possible additional cause of irritation.

INFLAMMATION OF THE UTERUS.[1]—METRITIS.

By W. O. Priestley, M.D., F.R.C.P.

Like other organs in the body the uterus is liable to congestion and inflammation, and in its course the inflammation may be acute or chronic.

Congestion of the Uterus.—Although it may reasonably be doubted whether an organ simply congested can be described as in a state of inflammation, it is sufficiently established that congestion or hyperæmia constitutes the first step in the inflammatory process, and that but a slight interval, characterized by more intense action, separates congestion from inflammation, and the formation of those products which are usually associated with inflammation.

The uterus, from its anatomical relations and the peculiarity of its functions, is specially liable to congestion. In healthy women, as each monthly period comes round, there is a large influx of blood to the uterus and neighbouring parts, in connection with the periodic and regular maturation of ovules, or "spontaneous ovulation," as it has been termed. The production of the catamenial flow is the result, and after its persistence for a certain number of days the congestion subsides, and the parts return to their ordinary quiescence. Should anything occur to disturb the regular and proper performance of this physiological process, the uterine congestion, which within certain limits is normal and salutary, may readily be pushed beyond the confines of health, and become morbid. Thus, as the result of functional derangement, or of displacement, or of organic disease in the womb and its appendages, more than the due amount of blood may distend the vessels at the catamenial periods, and instead of the usual moderate menstrual flow, a discharge of blood occurs, which, both on account of its amount and persistence may be characterized as hæmorrhage. Or, as the result of the same class of causes, the loss of blood at the menstrual periods is not only abnormally large, but the recurrence of what should be the periodic flow becomes irregular and too frequently repeated. Again, if only the normal amount of blood is sent to the tissues of the uterus and any cause prevents the proper elimination and secretion of the catamenia, by which the congestion is relieved, the uterus will remain large in bulk, and increasing as each month returns, by a repetition of the same events, it will gradually become hypertrophied.

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[1] This article was written in 1872—73.

The consequence, therefore, of persistent or frequently repeated attacks of morbid congestion is that the uterus becomes permanently enlarged, and as its substance is composed of dormant elements readily developed into more mature structures, the steady influx of blood eventually gives rise to a genuine hypertrophy. Besides the increase in bulk the tissue then becomes harder and denser and more resisting to the knife. According to Virchow this condition is due to increase in the amount of connective tissue, and Dr. Thomas consequently proposes to give it the appellation of "chronic hyperplasia," to distinguish it from inflammation. Scanzoni, on the other hand, believes that the muscular element is also augmented.

This hypertrophy may affect equally the body and cervix of the womb, the whole organ becoming larger and heavier; or it may affect a limited portion, as one or both labia uteri, singly or together. The cervix has been observed so equably enlarged in its lateral dimensions, as to form a considerable tumour in the vagina, and press inconveniently on the rectum. In one remarkable form of this local hypertrophy which has recently attracted much attention and is not infrequent, the whole cervix is so enlarged downwards, without the appearance of other morbid change, as closely to simulate prolapsus uteri, the os uteri in some cases projecting beyond the vaginal orifice, while the fundus maintains its normal position in the pelvis. This form of hypertrophy has been specially described by Virchow and M. Huguier. Closely allied is a form of uterine hypertrophy produced by defective involution of the uterus after delivery. In this, the process by which the womb is reduced to the size of the unimpregnated organ is suspended or arrested, and the organ remains large, heavy, and relaxed, with its cavity elongated and its vessels over-distended. It has been supposed that the supervention of puerperal inflammation in the early days after childbirth is concerned in the production of this condition, and no doubt this is so in some cases, but in a large number of instances it occurs where there has been no history of preceding inflammation, and where the fault seems to have arisen from imperfect contraction of the uterine fibres, combined with feeble reparative power peculiar to the constitution of the patient. Such a state of the uterus exists more frequently after abortion than after delivery at the full period, and it can readily be understood that the weakness of its tissue predisposes to uterine congestion and inflammation, while the increase in size predisposes to displacement.

Dr. Thomas, who has devoted much intelligent consideration to this subject, believes that a large proportion of cases called chronic parenchymatous metritis are really later stages of sub-involution; and that the persistent hyperæmia present in the early stage of this condition engenders "hyperplasia," or increase of the connective tissue as a result of a vice of nutrition without really partaking of the character of an inflammatory process. Persistent hyperæmia arising from other causes may possibly produce a like hyperplasia in the nulliparous uterus, but such a result is infrequent. When, however, the uterus has once undergone the development of pregnancy, areolar hyperplasia may be induced at any time, by displacement, endometritis or by other conditions producing continued hyperæmia, without the previous existence of sub-involution.

Acute Metritis.—Acute inflammation may attack any of the three coats of the uterus, or all conjointly.

1. *Serous or Peritoneal Coat.*—When the serous or outer covering is inflamed, it is generally by extension through the structures beneath, or by continuity from the broad ligaments on each side. In either case the affection becomes one of the forms of *pelvic peritonitis*, and the reader is referred to the article on that subject.

2. *Middle or Fibrous Coat.*—Acute inflammation of the parenchyma of the uterus is met with in its most characteristic form in connection with the puerperal state. Occurring after delivery, it is, however, in most cases rather the localization of a constitutional affection; general blood-poisoning preceding or at least being intimately associated with it. The pathological results observed are intense hyperæmia, swelling and œdema, with softening of the tissues of the uterus. The os and cervix uteri commonly appear as if contused and ecchymosed. When the morbid action has been great and the attack sufficiently prolonged, the uterine cavity may contain pus mixed with the lochia. The inner surface of the organ presents a deep livid or purple colour, particularly at the placental spot, where shreds of decidual and uterine tissue appear almost gangrenous. The middle coat is thickened, soft and friable and infiltrated with blood and pus; and the peritoneal surface and broad ligaments are covered with a low form of lymphy exudation, probably mixed with imperfectly-formed pus. The dilated blood vessels are frequently observed to have their coats inflamed, and to contain coagula in all stages of disintegration mixed with pus. The lymphatics, even more distinctly, may be seen distended with pus, and in some cases may be traced beneath the peritoneum, from the uterus to the broad ligaments, and thence upwards to the larger lymphatic ducts and glands lying in front of the vertebral column. Graphic illustrations of these morbid conditions are to be found in Cruveilhier's "Morbid Anatomy."

Virchow has described a puerperal form of inflammation, which he designates "diffuse puerperal metritis," and which prevails when epidemic influences are in operation, the whole affection bearing the greatest possible resemblance to erysipelas and pseudo-erysipelas on the surface of the body.

In the unimpregnated uterus, acute inflammation of the parenchyma, of the idiopathic form, is comparatively rare. The modern practice of treating many affections of the uterus by surgical proceedings, some of them being of a grave and severe character, has considerably increased the frequency of what may be termed acute traumatic inflammation. Thus attempts to enucleate fibroid tumours and extensive divisions of the cervix, have been followed by acute parenchymatous metritis and peritonitis, in some instances ending fatally. A peculiarity deserving notice in reference to these results of operations on the uterus is, that the inflammatory attack does not always immediately succeed the operation, but often lies dormant for many days, and then without appreciable cause suddenly develops itself. Thus not infrequently a patient has seemed to progress favourably for five, six, or more days, after an operation on the uterus, when suddenly acute inflammatory symptoms burst forth, giving all the evidences in a short time of a matured attack of metritis or metro-peritonitis. The explanation is probably to be found in the inactive condition of the chief elements composing the uterine walls in the unimpregnated condition. This necessitates the lapse of a certain time to develop the phenomena of inflammation, but when once a certain momentum is attained, the morbid changes go on rapidly, and as the peritoneal covering becomes involved, the pain and tenderness give unequivocal evidence of the nature of the attack. Or it may be that the inflammatory action slowly passes along the mucous coat, through the uterine cavity and Fallopian tubes, until it reaches the peritoneum, and then declares itself by prominent signs.

The practical deduction is that operations involving deep incisions into the uterine substance should not be lightly undertaken, and in those cases where absolutely necessary, the patient should be kept perfectly at rest for a sufficiently long period to ensure immunity from inflammation; further the practitioner should not be lulled into a false security against untoward consequences because no indications of mischief present themselves during the early days succeeding the surgical procedure.

The inflammation in these cases is rarely limited to the uterus itself. Most frequently it is complicated both by peritonitis, ovaritis, and cellulitis, and if the affection attains any considerable intensity the womb speedily becomes fixed in the pelvis, partly by the lymphy exudation on its peritoneal surface, which glues it to neighbouring structures, and partly by lymph poured out into the meshes of the cellular tissue of the pelvis. The adhesions thus formed often assume more or less of a permanent character, and by thickening the *indusium* of the ovaries and distorting and occluding the Fallopian tubes, may interfere with the functions of menstruation and conception; or by fixing the uterus firmly in the pelvis prevent its proper expansion should conception occur, and thus lead to inevitable abortion. Abscesses have been found in the walls of the uterus after acute inflammation, but these are generally of small dimensions, the larger abscesses found associated with metritis being generally situated in the pelvic cellular tissue.

3. *Mucous Coat.*—Acute inflammation of the mucous-membrane of the uterus or acute endo-metritis is commonly the precursor of inflammation of the other coverings of the organ, and this probably because

it is more accessible to external agencies and injuries. It is always present when the parenchymatous or middle coat of the womb is inflamed, and this occurs chiefly in puerperal patients, but acute endo-metritis or inflammation of the mucous membrane occurs somewhat frequently in the non-puerperal uterus. In these cases, the sub-mucous layer of connective tissue and the strata of the contractile coat which lie nearest to the mucous surface are commonly more or less affected, but the inflammatory process may not penetrate deeply into the thickness of the middle coat, unless action is very intense. Acute endo-metritis is not uncommon in the progress of exanthematous fevers, giving rise to profuse and painful leucorrhœa, and probably remaining after the fever has passed, in the form of chronic endometritis. This has been called by Kiwisch " metastatic constitutional catarrh." It may occur also as the result of a sudden check to the progress of menstruation. Perhaps the most frequent form in the unimpregnated uterus is that produced by gonorrhœa, which has extended upwards from the vagina and urethra. The morbid process may affect only the cavity of the cervix, or it may run along into the proper uterine cavity, traverse the Fallopian tubes, and eventually light up pelvic peritonitis. When menstruation is suddenly checked inflammation commences in the interior of the womb. In puerperal cases it probably has its starting point also in the uterine cavity, and in the vicinity of the placental spot. When provoked by gonorrhœa, or it extends in any way from the vagina, it begins at the os uteri and makes its way upwards. The effect of acute inflammation is to produce intense redness of the mucous membrane, and the hyperæmia is followed by softening and œdema. It is easier then than in the normal state to separate the mucous from the subjacent layer, and some portions look rough and ragged as if partly exfoliated. Scanzoni states that the surface is often spotted with bright red, the red places corresponding to the artificial openings of the utricular glands, which are surrounded by a fine capillary network very strongly injected. The uterine cavity generally contains an undue quantity of liquid, consisting of mucus or muco-pus, sometimes reddened by blood, sometimes like cream. There is usually more or less congestive swelling of the muscular coat of the womb at the same time, and the layers next the mucous membrane are gorged with blood. Here there may be the infiltration and softening characteristic of parenchymatous metritis, and it extends the deeper in proportion to the intensity of the morbid action on the mucous surface. Klob says the whole substance of the uterus appears to be increased. The same changes are observed in the canal of the cervix, though perhaps in a less marked degree than in the body. The os looks tumid, its labia puffy, and the papilla, deprived of their epithelium, stand out, producing the appearance of a granular abrasion.

Chronic Metritis.—Chronic inflammation of the uterus differs from the more acute form, not only in its less rapid progress and the less severe symptoms by which it is accompanied, but it differs also in reference to certain pathological results which are observed in association with the tardier morbific process.

1. *Peritoneal Coat.*—Chronic inflammation of the peritoneal coat of the uterus is comparatively of less pathological importance than chronic inflammation of the other coats, and probably rarely occurs except as a secondary result of inflammation in the adjacent structures or as an extension of a more general peritonitis. The results observed consist chiefly of lymph exudations on the free surface of the membrane, occasionally thrown out so copiously as to glue the various appendages of the uterus together, and to produce adhesions between the uterus and large and small intestines. A sufficiently common illustration of this effect is familiar to the uterine pathologist in the frequently observed union between the posterior surface of the uterus and the front of the rectum, the adhesion being so close in some cases that separation is impossible, and the functions of both organs are alike impeded by the union, or are performed with difficulty and discomfort. Occasionally, too, in cases of retroflexion, the fundus uteri becomes adherent to the anterior surface of the rectum, and the replacement of the womb, either by the finger or sound, is thus prevented so long as the adhesions exist. It is a curious fact that these adhesions are often found when there is no distinct history of previous acute inflammatory symptoms.

2. *Middle Coat.*—Chronic inflammation of the middle coat or parenchymatous structure of the uterus is generally an extension from the mucous coat and produces thickening and induration of the tissue, bearing some sort of relation to the duration of the morbid process and the amount of inflammatory action. This condition differs somewhat from the slower hypertrophy which arises from chronic hyperæmia, and has been called "areolar hyperplasia" by Dr. Thomas, inasmuch as it is attended by inflammatory exudation into the parenchymatous structure, and may, in some cases, give rise to considerable intumescence, produced more speedily than chronic hyperplasia, and more amenable to subsequent removal. The immediate effect is the infiltration of liquor sanguinis into the meshes of the uterine structure. At first the whole substance of the coat is softer and less resisting from the fact that not only is the interstitial deposit plastic in its character, but the elementary fibres are themselves softened by the inflammatory action. After a time a certain shrinking and solidification succeeds, by which the parenchyma becomes much firmer than in health, and if the process be continued or the attack often repeated, the whole thickness of the walls becomes permanently increased outwards, by the growth of connective tissue, as in hyperplasia, while from the shrinking afterwards, the cavity becomes elongated and enlarged after the fashion of the ventricles in eccentric hypertrophy of the heart. The hypertrophy produced by chronic inflammation may be general, producing an equable enlargement of the entire womb, or it may be partial and limited, affecting only a portion of the organ. Sometimes the fundus only has its bulk

increased, and as the effect of its greater weight, it may fall forward upon the bladder or backwards upon the rectum. This is not invariably the result of enlargement of the fundus, but when it does occur hypertrophy becomes complicated with displacement. The commonest form of partial hypertrophy met with in practice, so far as the supra-vaginal division of the uterus is concerned, is an enlargement or undue prominence of the posterior wall, the projection being so great in some instances as to have led Dr. Oldham [1] to suppose that it was frequently mistaken for retroflexion of the womb. More rarely the enlargement takes the form of masses which project as nodules of variable size from the outer surface of the uterus, and may be distinctly felt through the abdominal walls. Instances have been observed in which the irregular inflammatory deposit on the outer surface of the uterus reached the bulk of a large orange, but having a more irregular and nodular outline, simulating in form and relations some of the varieties of fibroid tumour, but obviously not of that nature, as proved by its complete disappearance under treatment. In these cases it is probable that some of the swelling is produced by simultaneous inflammatory exudation into the sub-peritoneal cellular tissue which surrounds the middle coat of the uterus, and this conjecture receives confirmation from the fact, that large inflammatory tumours of this character are more commonly observed towards the lateral borders of the organ, where cellular tissue is more abundant than elsewhere. Abscess in the parenchyma of the uterus, produced by chronic metritis, is extremely rare.

The cervix, however, is principally or exclusively the seat of enlargement, from chronic inflammation in the largest number of cases. Occasionally one lip only is affected, in which case it becomes broader, more prominent, and projects lower into the vagina than the other. In other instances the whole vaginal portion of the cervix is implicated, both labia being extended in breadth, and being considerably elongated. In women who have borne children and in whom the os uteri may have been fissured during parturition the hypertrophy may be more or less lobulated in its character. It may be remarked that in the lobular or irregular hypertrophy of the cervix which has been produced by simple inflammation, the intervening sulci or fissures commonly radiate from the os uteri as a centre, while in contra-distinction to this, the lobulation of the cervix in the earlier stages of malignant disease, is, as a rule, more irregular, the sulci having no such definite arrangement or tendency to converge to the os tincæ. A form of hypertrophy of the cervix with lobulation, in the production of which, chronic inflammation probably plays an important part, is that in which mucous cysts constitute a large portion of the projecting mass. In some of these cases, not only is the muscular structure and connective tissue of the cervix increased to a large amount, but the mucous follicles or glandulæ nabothi undergo an abnormal development, and the superficial ones appear as firm opalescent elevations on the surface. Ordinarily, these kinds of hypertrophies are of limited extent, but instances are occasionally met with in which they grow to such an extent as to form considerable vaginal tumours, and from the irregular character of the lobulation are liable to be mistaken for malignant disease.

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[1] Guy's Hosp. Reports.

It may be difficult to determine in some of the forms of enlargement above described, whether they are the genuine products of inflammation or only the results of chronic and persistent hyperæmia. It is quite certain, however, that the development of some of them is preceded by exudation and is accompanied by symptoms which overstep those of mere persistent congestion, and yet which cannot be described as those of acute inflammation.

3. *Mucous Coat; Chronic Endo-metritis.*—At one time authors on uterine diseases commonly described the inflamed uterine mucous membrane as prone to the exudation of coagulable lymph on its free surface. It is now well known that the production of inflammatory exudation on the inner surface of the uterus, although not uncommon in the more acute attacks of metritis, associated with the puerperal state, is rare in the unimpregnated condition. The observations of Dr. Oldham, and later, those of Sir J. Y. Simpson, have conclusively proved that certain membranous structures, called "dysmenorrhœal membranes," periodically expelled from the womb, and which were taken to be portions of coagulable lymph, being erroneously compared to lymph casts from the trachea and bronchi, are really exfoliations of the mucous membrane itself, shed off under certain abnormal conditions of menstruation. A minute description of these pathological products may be more appropriately given under the head of "Dysmenorrhœa," but a passing notice can scarcely with propriety be omitted here, since the process by which they are evolved, if not essentially inflammatory, bears a close relation to it. The membranes thus expelled consist of shreds or patches of variable size and thickness, in accordance with the extent and depth of the exfoliation. Occasionally the whole superficial layer of the mucous membrane of the cavity of the womb is separated in its entirety, and represents a triangular cast of the interior of the uterus. The structure of these membranes or shreds of membrane, is essentially the same as that of the lining membrane of the uterine cavity. The one side, representing the free surface of the membrane, is smooth, and is studded over with minute pits or depressions, which are the orifices of the mucous glands or follicles. The other side, which has been adherent to the uterine walls, is rough and flocculent, as the result of the process by which it has been torn off or detached.

In any case where the uterus forms and expels these deciduous membranes, the function of menstruation is performed with much discomfort, and the spasmodic pain which attends their extrusion towards the end of the catamenial period, is sometimes so severe, as to be compared to the throes of labour. During menstruation under these circumstances, the uterus grows abnormally heavy, and is very tender to pressure. It gives a sense of increased temperature to the touch, and its arteries can be felt beating with undue impulse and frequency. The more acute symptoms gradually subside with the departure of the period, but frequently there is more or less of *malaise* prolonged into the interval, and the womb is then commonly found more or less habitually turgid, and its tissues indurated by interstitial exudation.

Dr. West believes these so-called "dysmenorrhœal membranes" can scarcely be inflammatory products, inasmuch as the exaggerated vascular changes by which they are accompanied at the catamenial periods leave but slight changes indicative of inflammatory action in the intervals. On the other hand it may be doubted if the womb, habitually shedding these membranes, is ever quite free from some congestion or induration, and slight though this may be, it is commonly perceptible on examination. Scanzoni, indeed, recollects but a single woman affected with membranous dysmenorrhœa, whose uterus was not the seat of chronic engorgement, fibroid tumour, or flexion.

The late Dr. Edward Rigby believed these structures to be produced as the secondary effect on the uterus of some morbid change or irritation going on in the ovaries.

4. *Uterine Catarrh; Uterine Leucorrhœa* or *Metrorrhœa.*—This affection, which is characterized by a superabundant flow of mucus, or muco-purulent discharge from the uterus, may more accurately be described as one of the symptoms of endo-metritis than as a special affection by itself. It bears the same relation to inflammation of the uterine mucous coat as catarrh of other mucous surfaces does to inflammation there. It is commonly chronic in its character, but, as before stated, cases of acute endo-metritis attended by catarrh are not unfrequent; that produced by gonorrhœa being a familiar example, and hence, various authorities have described both an acute and chronic form of uterine catarrh, the one differing from the other only in the greater or less intensity of the associated symptoms. Many gradations may be observed between the two extremes; the more acute, as the more active symptoms subside, passing into the chronic form, and chronic endo-metritis, under some fresh stimulus to inflammatory action, readily lighting up into the more acute type.

The pathological changes commonly observed in the mucous membrane are much the same as in acute endo-metritis, but less in degree; all its capillaries become injected, and its surface presents a deep red colour. The various structures soften and swell from effusion, and patches of ecchymosis may be seen here and there from the rupture of minute blood-vessels into its substance. Occasionally ulcerations have been found in the proper uterine cavity as the result of internal metritis, but these are probably rare in their occurrence. Erosions and pseudo-ulcerations are produced with comparative frequency in the cervical canal. The usual result of this perverted action is that the follicular structures become hypertrophied and the secretion of their mucus is largely increased in quantity and its characters are in some respects materially altered. The amount and quantity of the discharge vary with the stage of the inflammation and with its intensity, and the character of the secretion differs also in accordance with the portion of the uterine cavity by which it is furnished. Thus, in the early stage of a more acute attack, when vascular excitement is at its highest, all secretion may become checked, and it is not until the climax is passed that muco-purulent secretions are poured out in greater or less abundance, the appearance of the discharge being generally coincident with an amelioration of suffering. The discharge flowing from the uterine cavity is commonly muco-purulent, sometimes tinged with blood, and occasionally, truly hæmorrhagic. Dr. Henry Bennet has drawn especial attention to the distinctions between inflammation of the uterine cavity proper, and inflammation of the cervical canal. The latter (endo-cervicitis) he believes to be a frequent affection, while the former is comparatively rare. Commonly the discharge furnished by the upper part of the uterine cavity proper is comparatively thin and limpid, while in contradistinction, that from the gland follicles of the cervical canal is gelatinous and viscid, this difference in the thickness of the secretion furnishing, in some sort, the clue to the locality from which the discharge comes. Dr. Tyler-Smith and Dr. Hassall have pointed out that the mucous membrane lining the cervical canal is furnished with a most elaborate secretory apparatus, which they have carefully delineated, and it is the peculiarity of the gland follicles in that locality to secrete a thick viscid mucous which forms a plug to seal the os uteri in normal pregnancy, but which may also be poured out in undue abundance from disease in the unimpregnated condition. Dr. Henry Bennet avers that the discharge from the uterine cavity in endo-metritis is often more or less mixed with blood so as to have a rust-coloured appearance, especially for a fortnight after menstruation. This he looks upon as being "as characteristic of internal metritis as the rust-coloured expectoration is of pneumonia," —a statement requiring some qualification, as it is not by any means a constant sign. As pointed out by Dr. Thomas, there is a variety of corporeal endo-metritis, sometimes involving also the vagina, which occurs in old women, after menstruation has ceased, in which a watery or creamy pus is secreted, which is very irritating, and a like affection is observed sometimes in younger women. The secretions of the uterine cavity have ordinarily an alkaline reaction, and thus if clear and pellucid as they ooze from the os uteri, they become opaque by the admixture with the acid mucus of the vagina. Both the alkalinity of the uterine secretion and the acidity of the vaginal mucus are apt to be exaggerated by the inflammatory process, and Donné avers that in either case spermatozoa are killed after coitus. Hence sterility is observed with existing inflammation of the mucous membrane lining the genital tracks, and pregnancy occurs when it is cured. As in the more acute attacks, the parenchymatous structure rarely escapes participating in the inflammatory action, so even in the more chronic forms, induration and thickening of the uterine substance is frequently associated with the morbid changes in the mucous membrane. The cavity then becomes expanded both in its lateral and longitudinal dimensions; the os internum widens, and the sound passes more easily, and, it may be, penetrates an inch or more deeper than ordinarily. There is, however, a large class of chronic cases in which but comparatively little alteration is noticeable in the deeper uterine structures, and yet where the catarrh is both profuse and obstinate. In most of these instances the catarrh is from the cervical canal. There the labia uteri are usually more or less tumid and pouting, and the os externum is expanded, the whole cervical canal being larger, and filled with a dense mass of adhesive mucus which projects from the os uteri and is dislodged with difficulty. Occasionally cervical catarrh is associated with marked stricture of the os and cervical canal. When the inflammation is confined to the cavity of the cervix, the os uteri internum retains the contraction which is found under normal circumstances and the sound encounters the usual obstruction there. Great circumspection is however necessary in the use of the sound, as the uterine cavity when inflamed is commonly very sensitive, and the mucous membrane often bleeds profusely when touched by a probe or other instrument. The catarrh is often associated with abrasion or granular inflammation around the os uteri, the discharge by its acrid property irritating the mucous membrane there, and causing it to shed its epithelial covering. The frequency of abrasion in conjunction with uterine leucorrhœa induced M. Huguier and Dr. Tyler-Smith to advance the hypothesis (which will be again referred to), that abrasion and ulceration of the os uteri are mainly due to hypersecretion from the cervical cavity. The presence of abrasion around the os uteri in cases of uterine catarrh is nevertheless by no means constant, instances being frequently observed where it is absent, notwithstanding an abundant hypersecretion habitually poured from the uterine orifice.

5. *Abrasion, Granular Inflammation, Erosion, Ulceration,* of the mucous coat.

These conditions, which commonly affect the mucous membrane round the os uteri, have been the occasion of much debate among uterine pathologists, and great diversities of opinion have been expressed, both in reference to their exact definition, their nature and character, and also concerning their pathological importance. Dr. Henry Bennet, Dr. Tilt, and others consider all to be varieties of inflammatory ulceration, the terms abrasion, granular inflammation, and ulceration representing a progressive series of pathological changes which reach their climax when completed. Other observers, among whom may be enumerated Dr. E. Rigby, Dr. Snow-Beck, Sir James Paget, and Dr. Arthur Farre, regard simple erosion and granular inflammation as having no claim to be classified as varieties of ulceration, and as possessing none of its essential characteristics, such as the loss of the vascular and parenchymatous tissues. Some authors again hold that inflammation and ulceration of the cervical mucous membrane have a primary importance at one time unsuspected, that, having mainly a local origin, they affect materially not only the comfort, but the general health of the subjects of them, are the parents of other diseases, and, as a natural corollary, demand chiefly local treatment.

In opposition to this view the evidence of prolonged and careful observation has been adduced to show, that an undue importance has been attached to simple ulcerations of the os uteri; and that admitting their frequency, in a large proportion of cases they give but little evidence of their presence, are attended with but slight local inconvenience, and are rather to be looked upon as complications of some more important ailment, or as evidences of some constitutional derangement, than as primary affections, having an essentially local origin. The contention concerning the definition of uterine ulceration has mainly taken place because an erroneous impression gained considerable currency, that the presence of ulceration once determined, frequent instrumental examinations and cauterization are necessary. It is however obviously undesirable to multiply terms and definitions because some practitioners persist in taking an overstrained view of the importance of these pathological changes, and pursue methods of cure which may be entirely uncalled for by the circumstances. Studied as pathological phenomena and apart from doctrinal views, the changes taking place in the mucous membrane of the os and cervix uteri, when affected by chronic inflammation, may generally be said to be the following:—The membrane becomes swollen and puffy, and the minute vessels beneath the surface are unduly injected. The last stage indeed is that of hyperæmia or congestion. If action goes beyond this, the squamous epithelial particles lying most superficially become cloudy and opalescent, and losing their cohesion, are shed off singly, or in patches to mingle with the mucus of the genital passages. The papillæ beneath, thus denuded of their outer covering, present a red velvety appearance, readily bleed when touched, and if nitrate of silver be applied are at once covered with a white film from the production of the chloride of silver. This effect is less noticeable where the epithelium is entire. These changes, which constitute *abrasion* or *erosion*, are observed immediately around the os uteri, in a ring of varying breadth, but they are sometimes seen on other parts of the cervix, and, not infrequently, groups of red and inflamed villi, denuded of epithelium, are seen scattered over the whole mucous membrane of the vaginal portion of the cervix, looking at first like numerous spots of acne, but eventually becoming isolated or confluent patches of abrasion. The erosions or abrasions thus produced are sometimes smooth and uniform on the surface, and then resemble those produced by mechanical attrition; at other times they are irregular and worm-eaten in appearance from the unequal way in which the epithelium has been exfoliated—bundles of filiform papillæ projecting on the surface, and small pits or depressions being readily discerned, caused by the epithelium being emptied out of the mucous crypts and follicles. The abrasion when limited to one uterine lip is, in the largest number of cases, but not exclusively, situated on the posterior one, and whether partial or completely encircling the os uteri, commonly is associated with some dilatation of the orifice. When the labia uteri are separated the erosion is seen to run up the cervical canal as far as it can be examined, giving rise to copious muco- or muco-purulent discharge—being complicated in fact with inflammation of the cervical canal and catarrh. Various eruptions analogous to those observed on the skin have been described as associated with these simple abrasions. Thus acne, herpes, and lepra have been mentioned as occurring on the cervix uteri, and these in the later stages of their progress leave erosions of the mucous membrane. The best recognised eruption in this locality is the herpetic form, in which one or several flattened vesicles form on the surface of the previously congested mucous membrane, and eventually burst, leaving abrasion beneath. Herpetic vesicles are apt to be mistaken for mere chronic distensions of the glandulæ nabothii, several of which sometimes co-exist, varying in size from a millet seed to a pea, and project irregularly from the surface of the cervical mucous membrane. In some instances the epithelium is detached in small patches, the intervening tissue being healthy or only somewhat redder than natural. The mucous membrane then seems beset with minute aphthous ulcerations, similar to those observed in the mouth and elsewhere. MM. Boys de Loury and Costilhés, M. Robert, Drs. Tilt, Matthews Duncan and others have described a diphtheritic or pseudomembranous exudation of the cervix uteri and vagina, which is usually accompanied with superficial inflammation of the parts, and which, on removal, leaves loss of epithelium and abrasion beneath. Under the microscope these membranes are found to consist of delicate fibrillæ and epithelium, and they are bounded by a red inclosing line with elevated margins, which makes them easily mistaken for deeper ulcerations.

Should hyperæmia proceed beyond the production of mere abrasion, *granular inflammation* or *ulceration* follows. The erectile papillæ having lost entirely or in great part the epithelial covering which normally binds them down, and having received fresh impetus to growth, present a vivid red colour, and an appearance of prominent granulations. These granulations are either on a level with or project somewhat above the general surface, and are bounded by the surrounding sound mucous membrane, which either preserves its natural colour or assumes a somewhat deeper red in the immediate vicinity. Occasionally the granulations are so exuberant as to assume a fungoid appearance. They then readily bleed, are probably more or less less sensitive to the touch, and have a slightly indurated raised border, with a vivid red boundary line running just within it. This condition, if persistent, is nearly always associated with some degree of thickening in the subjacent structure of the cervix, and hence the labia look puffy and swollen, and often deep sulci radiating from the uterine orifice divide them into lobules, in such a way as to make the granulations appear still more prominent. One of these varieties of exuberant granulation has been termed "cock's-comb" granulation from the supposed resemblance to a cock's-comb. Dr. Arthur Farre has pointed out that one effect of inflammatory action is to produce eversion of the hypertrophied mucous membrane in the cervical canal, and that this is apt to be mistaken for a more genuine form of ulceration. In these cases the apparent unevenness and rawness of the surface, the prominence of the rugæ, and the recession outwards of the natural boundary line forming the os uteri externum, all conduce to produce the appearance of ulceration. Somewhat more rarely occur those forms of ulceration in which the surface is depressed below the general level of the mucous membrane. Instances of distinct excavation with loss of the deeper structures as the result of simple inflammatory action are not frequently met with when the uterus is *in situ;* but in prolapsus, where the cervix is habitually exposed to external agencies, and kept constantly dry, it is by no means uncommon to find around the os an excavated ulcer so well defined and limited, that it might have been dissected out with a knife.

When any considerable thickening of the labia uteri takes place as the result of persistent hyperæmia associated with the ulcerative process, the microscope shows that there is not only hypertrophy of the contractile structures, but an especial increase of the connective tissue. This gives a greater density to the cervix, and if the closed follicles or glandulæ nabothii become distended and project at intervals over the surface, as occasionally happens, a very marked feeling of irregularity is observed on making a digital examination. The partial hypertrophies assume a diversity of forms. In one often observed, there is a projection varying in size from a split pea to a cherry, on the inner side of one of the labia towards the os uteri. If the opposite lip then remains sound, it is thinned out and spread over the nodule. On the most prominent point there is usually marked erosion, while elsewhere in the vicinity there is much vascular injection, and copious muco-purulent or sanguineous discharge is poured out from the surface.

The general colour of the cervix affected by these morbid changes varies exceedingly. Sometimes the mucous membrane surrounding the inflammatory patch deviates but little in this respect from its normal appearance. It may indeed be pale and anæmic, affording evidence of feeble circulation and defective power. Occasionally, however, both ulceration and surrounding parts are of a deep violet or purple hue, like the appearance of the cervix and vagina in pregnancy; and there are further indications of intense venous congestion in a varicose condition of the smaller veins there. Hence the appellation of "varicose" ulcer of the cervix employed by French and other writers.

The varieties in the way of intensity, extent, and appearance presented by these pseudo and true ulcerations are so considerable, that attempts have been made to classify them, like surgical ulcers of the legs for example, but without any obvious practical advantage.

In some instances of chronic inflammation of the mucous membrane covering the cervix, no abrasion or ulceration takes place. The surface, without any breach of continuity, exhibits everywhere a deep red colour—has a finely granular appearance and is swollen and œdematous. In this condition the vaginal walls often take part, and the rugæ there become hypertrophied and prominent. The copious muco-purulent discharge secreted by the whole surface, and the pruritus and excoriation of the external parts from the acridity of the secretion, may raise the suspicion that it is gonorrhœal in character, more particularly as it may be the cause of a mild form of blennorrhagia in the male. The frequency of its repetition, however, in the same subject, the readiness with which it is reproduced by slight causes, its persistence in some patients even throughout pregnancy, and lastly its association with a gouty or rheumatic diathesis or with the various forms of dyspepsia and with acne on the face, all point distinctly to a constitutional origin. This variety of chronic inflammation is frequently observed in women who have passed the climacteric period of life.

As to the way in which abrasions and ulcerations are produced, and the order of the several pathological phenomena, various hypotheses have been advanced. One school of authorities, of which Dr. Henry Bennet is the able exponent, holds that when the uterus is the seat of chronic inflammation, the os and cervix are the *points de depart*, and the result of that inflammation is ulceration there, attended with muco-purulent discharge, and hypertrophy later. M. Huguier and Dr. Tyler-Smith believe that the canal of the cervix uteri is the more frequent seat of inflammatory action in the first instance. Dr. Tyler-Smith maintained that it is here primarily that the phenomena of hyper-secretion and inflammation are most commonly observed, and that the flow of the perverted mucus from the os uteri produces abrasion and ulceration in the surrounding parts, these later changes therefore being secondary to the affection of the cervical canal. Dr. West in his admirable "Lectures" considers both these theories as expressing a too partial view of the subject, and urges that if any part of the womb has a pathological importance above the rest, it is the mucous membrane lining the true cavity of the womb. It is this that undergoes the greatest changes during the whole child-bearing period of life, and it is the condition of this portion which at all times influences the rest of the womb more than any other part. In the majority of inflammatory uterine ailments, "it is the interior of the uterine cavity which suffers first; it is thence that the hæmorrhages are derived; thence that the greater part of the leucorrhœal discharge is furnished; and it is the irritation of that part of the organ that leads to the increase of its size, so apparent in the large proportion of cases of long-continued uterine ailment." Dr. West indeed holds that as a general rule there is no proof of the point of departure of the mischief being in the neck of the womb, more than its orifice, or appendages. Dr. West's investigations have done much to prove the comparatively small importance of abrasion and ulceration of the womb *per se;* and that when these are attended with any serious results, there is generally some superadded morbid condition of the cavity or walls of the womb, which is more directly concerned in their production. After the first introduction of the speculum uteri, by means of which practitioners were for the first time exactly informed concerning the presence and frequency of these ulcerations of the os and cervix, it was perhaps very natural that their importance should be overrated, and it was supposed discoveries had been made enough to account for all the hitherto obscure forms of uneasiness in the region of the womb. The progress of time, and the accumulation of more accurate observations, have since done much to moderate this estimate, and confine it to more precise limits. It is now very generally conceded that but little importance is to be attached to the slighter forms of erosion or ulceration of the cervix in the majority of cases; that when present without other complication, they rarely exercise any considerable influence on the patient's health and comfort, but are rather indications of feeble power, or are superadditions to some other pathological change. It is equally admitted, that some of the severer forms of inflammatory ulceration, beginning it may be as local ailments, and particularly when associated with hypertrophy and induration, are sufficient to cause much suffering, and may seriously impair the general health. In some instances, indeed, a slight and superficial abrasion becomes the seat of very undue sensitiveness, rendering the exercise of the generative functions painful. These cases are nevertheless exceptional, and in patients the subjects of them, there are generally other points in the uterine substance, where no abrasion exists, almost equally painful to the touch.

Under the appellation of "irritable uterus" the late Dr. Gooch described a condition in which the womb was so exquisitely tender and so sensitive to motion, that the slightest touch or movement of the body produced distress, and yet on examination the organ was found unchanged in structure. As the result, the patient became "reduced in flesh and health, almost living on her sofa for months, or even years, from a constant pain in the uterus, which rendered her unable to sit up and take exercise." The proclivity of patients so suffering to take large and increasing doses of opium rather aggravated the malady, and tended to make it incurable.

It has been suggested that cases of "irritable uterus" are really cases of chronic inflammation in which the organic changes are less obvious than usual. Dr. Graily Hewitt again believes that some of Dr. Gooch's worst cases were instances of uterine flexion. The natural history and results of examination point rather to its being a form of uterine neuralgia, and Dr. Gooch and Sir Charles Locock noticed that it was habitually associated with a rheumatic or gouty diathesis.

Etiology.—The *causes* of acute metritis, besides those associated with the puerperal state, are sudden suppression of the catamenia from exposure to cold, either atmospheric or in the form of cold baths; the injudicious use of vaginal injections at unseasonable times or when too strongly medicated; the action of certain emmenagogues; operations on the uterus, and the use of strong escharotics; the incautious use of the uterine sound, and the employment of intra-uterine pessaries and expanding tents; physical injuries, and too frequent coitus, or sexual intercourse accompanied by undue violence. The first attempts at coitus after marriage in some sensitive women have been known to produce it. Again, the extension of gonorrhœal inflammation from the vagina may involve the uterus, Fallopian tubes, and ovaries, and light up acute inflammation there, and in this form the action is commonly met with in great severity. Lastly, the inflammatory action may originate in the ovaries, and pass by sympathy and contiguity to the womb itself.

The causes of uterine congestion and chronic inflammation may be described as indirect and direct. Under the first head may be classed natural feebleness of constitution; unhealthy conditions of life; impoverished conditions of the blood; the various ailments which obstruct the venous circulation above, and lead to passive congestion of all the pelvic organs. Among these may be enumerated certain forms of disease of the heart, lungs, and kidneys; habitual derangement of the liver and chylopoietic viscera; constipation and abdominal tumours. Women who are the subjects of saccharine diabetes seem particularly prone to uterine congestion. The direct causes are—any agencies which produce partial or entire suppression of the catamenia, or other derangement of the menstrual function; the various accidents associated with abortion and delivery, and imperfect convalescence after parturition, with over lactation. The relation of sub-involution to congestion, hypertrophy, and inflammation has been discussed in a previous page. Fissuring and laceration of the cervix uteri during parturition frequently play an important part in originating and keeping up the several forms of inflammation of the neck of the womb. The bottom of the cleft long refuses to heal, and thus keeps up a constant irritation, which indurates and inflames the surrounding parts. Too frequent indulgence in sexual intercourse, or coitus at unseasonable times, are well recognised as potent causes of these affections. As before remarked, the first attempts at intercourse after marriage in some sensitive women are sufficient to light up a sharp attack of endo-metritis, which, as it subsides, remains as a chronic cervical catarrh for months, or perhaps years, after its inception. It has been remarked that women with whom attempts at sexual intercourse are frequent but imperfectly performed, or who live habitually in sterile marriage, are very frequently sufferers from uterine congestion and inflammation. Habitual masturbation will likewise produce the same results, and the womb is often greatly increased in size from this cause. At the climacteric period of life, when menstruation is becoming irregular, the uterus is sometimes so considerably enlarged as to favour the suspicion that fibroid tumour exists, and the diagnosis between the temporary engorgement and fibroid growth is most difficult. The later subsidence of the swelling, however, eventually clears up the doubt.

The presence of organic disease in the uterine walls is also a direct incentive to uterine congestion. Fibroid tumours, polypi, and cancer cause a larger determination of blood towards the womb, and local irritation there. This is especially the case at the menstrual periods, and hence the proclivity to effusion and hæmorrhage then. Congestion and inflammation of the ovaries provoke hyperæmia in the uterus, and just as the initial physiological act of menstruation is commenced in the ovary, and thence is propagated to the uterus under normal conditions, so certain marked changes taking place in the ovaries which simulate the phenomena of menstruation, lead ultimately to congestion of the uterus, which may terminate either in enlargement of the organ, or in hæmorrhage, or in both. In prolapsus uteri retroflexion, and anteflexion, the flow of blood is not only likely to be impeded in its passage through the womb, but the local irritation to which the organ may be subjected on account of its displacement or distortion, favours a constant plethora of its vessels, and thus entails all the symptoms and effects of uterine congestion. To the above causes may be added the effect of specific venereal poisons. Chronic inflammation of the uterine cavity and catarrh have seemed to owe their origin in some cases to coitus with a male imperfectly, though apparently, cured of old gonorrhœa. Lastly, over meddlesome treatment with speculum, sound, and the various instruments and pessaries employed in uterine ailments, must be enumerated among the causes.

SYMPTOMS AND PROGRESS.—An attack of acute metritis is commonly ushered in by a rigor followed by increased rapidity of the pulse, flushed countenance, and hot skin. The patient complains of acute pain and tenderness in the hypogastrium. The pain extends backwards to the loins, and shoots into each ovarian region and down the thighs. There is an abiding sense of weight and bearing-down, and the desire to pass water is almost constant, while micturition is often extremely painful. The intestinal mucous membrane sympathises also in the disturbance, and often there is violent tenesmus with a catarrhal or dysenteric affection of the rectum. The catamenia, if present at the time, are probably suddenly suppressed, and the vagina is found on examination to be hot and dry. The uterine secretion, however, varies, and sometimes the discharge is hæmorrhagic ; at other times it is muco-purulent mixed with blood, and so irritating in its quality that it scalds the pudenda and adjacent parts. The uterus is commonly so painfully sensitive to touch, that a satisfactory internal examination is scarcely possible. It may, however, generally be determined that the uterus is the centre of the morbific action. The fundus is tender to external pressure, but ordinarily it is not so far increased in size as to be distinctly felt in the hypogastrium, unless it has previously been the seat of hypertrophy or fibroid tumour, or the attack has supervened on recent delivery. After abortion and delivery there is occasionally such rapid increase in size, by effusion into its meshes, that in the course of a few hours it can be felt as a considerable tumour above the pubes, and subsidence is sometimes almost equally rapid when action has ceased. Where the tissue of the uterus is not so lax, the augmentation in bulk is less notable. When vaginal examination is practicable the cervix is found turgid and painful to touch; it is somewhat below its natural position in the pelvis, and the arteries entering its lateral borders pulsate with undue prominence. The vagina is dry and hot, or bathed with irritant muco-pus. Frequently the sympathetic fever is accompanied by frontal headache, nausea, and vomiting, and in susceptible subjects, by a variety of hysterical symptoms. The hysterical symptoms are sometimes so prominent during an attack of acute metritis, that, unless great care be taken, the local condition may be overlooked. Making due allowance for the difference in constitution in individual patients, the severity of the symptoms will be in proportion to the intensity of the local inflammation and the extent of the tissue involved. The most severe and perilous cases occur in connection with the puerperal state, and in the worst forms the marked phenomena are not limited to the symptoms detailed above. As the malady progresses the peritoneum becomes involved and there is more general pain and tenderness. Eventually tympanites appears, accompanied by increased restlessness, delirium, and other indications of cerebral derangement. The tongue becomes loaded, dry, and brown; the pulse becomes feeble, rapid, and thready, and the skin grows cold and clammy. Eventually the prostration may amount to collapse and end in death. In some of those examples where inflammation either has originated in a recent wound, or where there has been a sudden and violent suppression of the catamenia, there is good reason to believe that many of the symptoms are due to blood-poisoning, as in puerperal cases, and that the metritis is only part of a constitutional ailment. It is worthy of remark, that in some of those cases which have a fatal termination, very little hypogastric tenderness is complained of throughout the attack, and hence the difficulty of diagnosis is increased. In a proportion of cases the disease is accompanied with symptoms much less violent than the above. There may be local pain, irritating and excoriating discharge, painful micturition, and some febrile excitement, but the active symptoms gradually subside, and the patient recovers, or indications of chronic metritis eventually establish themselves.

The symptoms of congestion and chronic inflammation of the womb are extremely variable, whether we consider them in reference to their constancy, degree of intensity, or the locality in which they present themselves. As in other forms of chronic uterine ailment, there is often no sort of relation between the discomfort produced or the impairment of function which takes place, and the extent of local injury. It is a matter of common observation that a very slight deviation in uterine health may give rise to very troublesome and obstinate symptoms, while on the other hand considerable organic changes of structure may take place, nay, malignant disease of the cervix may make considerable progress, without giving rise to any grave symptoms. The same inconstancy of symptoms, noticeable during the progress of pregnancy, is equally observable in connection with chronic uterine inflammation—at one time there is marked pain and uneasiness referable to the uterus itself, or to the organs in its vicinity, and to these may be added derangements of function and pains in other and distant parts. At other times there is an absence of all these concomitants, and it is only the presence of some single sign, such as persistent discharge or sterility, which attracts the attention of the patient. During the progress of any particular case indications of internal disorder may at one time be very prominent, at another quite in abeyance without any very marked alteration in the local condition to account for the change.

Commonly the patient experiences a sense of weight and discomfort in the pelvis, pain in the ovarian regions and back, probably shooting round the abdomen and down the lower extremities. The pain is ordinarily aggravated by exercise and relieved by rest in the recumbent posture. Dr. Churchill has noticed in several cases a peculiar pain in three different localities, viz., "in the symphysis pubis, at the point of the coccyx and along the sciatic nerve to the knee, which he should hardly have attributed to the congestion and erosion had it not been removed by curing them." With bearing down there is probably associated a feeling of pressure on the rectum and bladder, and a frequent desire to evacuate these organs is followed by an imperfect sense of relief or aggravation of the previous irritation. The rectum and bladder are most frequently involved when some increase in size or displacement of the uterus has taken place, and the symptoms are no doubt partly due to the effects of physical pressure. In the more occasional cases where such disturbance is present without decided enlargement or displacement of the uterus, it is probably reflected irritation—more rarely there may be actual extension of inflammation to the bladder or rectum. Constipation and painful defæcation are frequently observed, and the fæces are often coated with muco-pus as the result of the irritation transmitted to the intestinal mucous membrane. Hæmorrhoids are not infrequent, and are apt to become more troublesome at the catamenial periods. Occasionally diarrhœa is present instead of constipation, and small quantities of mucus are repeatedly discharged mixed with but small quantities of fæces. The irritability of the bladder is sometimes most distressing—day and night being alike disturbed by the desire to void the urine. The urine itself is often high-coloured and loaded with urates; occasionally it contains quantities of mucus and pus-cells. The pain in passing water and the frequent micturition may, indeed, readily mislead the practitioner into the belief that his patient is suffering from some primary disorder of the urinary organs instead of the uterus. The catamenia are either too profuse and too frequent, or too sparing and attended with pain, or perhaps suppresssd altogether. All the symptoms of the intermenstrual interval are apt to be more intense with the occurrence of the menstrual period. Often there is severe dysmenorrhœa with paroxysms of uterine colic, and in a certain proportion of cases the cessation of the menstrual flow is followed for several days by such acute and persistent pain as to lead to the suspicion of acute inflammatory action going on. The discharge is often mixed with clots which give pain during their retention in, and expulsion from, the uterus, and in the membranous form of dysmenorrhœa, shreds or more complete casts of the uterine cavity are extruded. With some patients, again, the return of the catamenia brings notable relief, and the days of their continuance are the only ones in which there is comparative comfort.

The exercise of the generative functions is often rendered so painful as to lead to its discontinuance, while in long-standing cases where all tenderness may have passed, patients sometimes complain that the sensibility of the nerves is obtunded and all desire extinguished. Sterility is a frequent, though not an invariable, consequence, and if conception does occur in such condition there is proclivity to abortion. In the intermenstrual intervals the vaginal discharges vary. In a small proportion of cases there is a diminished secretion of the ordinary mucus, and the genital canals are drier than natural. More constantly there is over-abundant discharge either of clear mucus, or mucus mixed with pus and occasionally with blood. These ingredients in varying combinations produce clear and transparent, white or yellow, brown or reddish discharges, in accordance with the degree and stage of the inflammatory process. When vascular granulations are present, coitus or mere locomotion may at any time be followed by a scanty or more profuse flow of blood, and in some cases even when the patient is at rest, there is a constant tendency to dribbling hæmorrhage during the whole menstrual interval.

When leucorrhœal discharge is thick and gelatinous it is characteristic of chronic inflammation of the cervical canal, and it is the product of hyper-secretion from the gland follicles there. Not unfrequently in endometritis, there will be no uterine discharge for a time, and then, after considerable pain, there will be a gush of muco-purulent or sanguino-purulent fluid which has been pent up in the inflamed uterine cavity.

All these discharges are sometimes so acrid as to excoriate the external parts, and obstinate pruritus vulvæ is often complained of, produced by the irritating quality of the secretions from the parts above.

The constitutional symptoms associated with chronic metritis are a deterioration of general health, loss of appetite, torpidity of the bowels, spanæmia, lassitude, and mental depression. In more aggravated cases, or in patients of nervous constitution, there are often indications of dyspepsia, nausea, sickness, and biliary derangement; headaches, neuralgia, sympathetic pains and swelling in the mammæ, pains in the loins; and to these may be added the developments of hysteria in its protean forms, which, if comparatively in abeyance during the interval, become aggravated at each catamenial period. Sir James Simpson and Dr. Todd, both pointed out the constant association of infra-mammary pain with uterine and ovarian derangement, and this reflected infra-mammary pain is more frequently under the left than under the right breast.

There is rarely much febrile disturbance except in connection with the pain at the catamenial periods, when the patient may suffer from congestive dysmenorrhœa. Then the pulse increases in frequency, the countenance flushes, and shivering may alternate with febrile symptoms. At these times sympathetic nausea and vomiting often render the condition of the patient most distressing, hysterical symptoms are apt to be prominent, and headache and lassitude remain after the more urgent symptoms have passed.

Now and then the general symptoms assimilate themselves very closely to the signs of pregnancy. There is darkening of the areolæ and pains in the mammæ, nausea, vomiting and other sympathetic phenomena. If the uterus be enlarged at the same time and menstruation deranged, an error in diagnosis is readily committed. Dr. Tilt regards the signs of pregnancy, without menstruation being suspended, in comparatively young women, as *prima facie* evidence of the existence of internal metritis. Many of the general symptoms above mentioned may be secondary in their character, and have been provoked by the inability of the patient, suffering from a local ailment, to take exercise and obey such natural laws as are essential to the maintenance of health, but in a large class of cases in which chronic inflammation has arisen, so to speak, spontaneously, and has not been consecutive of parturition or abortion, there has been, from the outset, some departure from a sound constitutional condition. The local affection is the consequence and effect of the general derangement, and attention to the general health is of the first importance in the treatment.

The functional signs being so variable and uncertain, physical examination becomes necessary, when an accurate diagnosis must be made, and this may be had recourse to with the less hesitation when the patient is married, and the symptoms are grave enough to demand it. Local examination is conducted with the fingers, aided, when necessary, by the speculum vaginæ and the uterine sound, and the exploration may be both by the vagina and rectum. In certain cases only will the uterus be so much enlarged from the results of chronic hyperæmia or metritis, apart from the presence of some other form of uterine tumour, as to permit the fundus to be felt by external examination of the abdominal walls. When the outline of the uterus cannot be made out, there may be acute tenderness on pressure over the fundus, and this tenderness, even when unattended by discharge, Dr. Routh states, is indicative of what he terms "fundal endo-metritis." Where, however, congestion and slow inflammatory action have been very persistent, or where attacks have been often repeated, or have supervened on sub-involution, the enlarged womb may perhaps be felt above the symphysis pubis,—particularly when the patient is thin, and the absence of adipose tissue in the abdomen facilitates the examination. Internal digital examination rarely fails to detect some increment in the bulk of the uterus whenever the parenchymatous structure has been long affected. The organ then feels heavy,—its structure more turgid and denser than natural,—and often the womb sinks below its normal level in the pelvis. In one form the organ is movable, its outer surface smooth, and its shape is altered somewhat by the swelling having rendered less distinct the demarcation between the body and the cervix. In other forms, some irregular nodulation or bulging in the body or fundus, is felt either by the vagina or rectum, or those partial hypertrophies of the cervix previously described are detected by the sense of touch. Where action is still in progress, there will probably be undue sensibility to touch and increased heat and dryness of the vagina. If there be erosion or ulceration, some indication of its presence will be afforded by the loss of perfect smoothness around the os uteri, and by the perception of either a velvety surface characteristic of abrasion or the rougher and more irregular conditions produced by the deeper forms of granular inflammation and ulceration. The os uteri may be widely patent or narrowed and distorted in shape by the tumefaction and lobulation of the labia. Dr. H. Bennet lays great stress on the patency of the external os uteri as an indication of the presence of inflammation of the cervical canal. There are, nevertheless, constantly recurring examples in which the os uteri is widely dilated from the effects of some previous delivery or abortion, and where no inflammatory changes are in progress. Where flexion or version are present as complications of metritis, the cervix may be displaced anteriorly or posteriorly in the pelvis, or laterally to either side. The alterations on the surfaces of the cervix, detected by the finger, may be further verified by the use of the speculum, and by means of this instrument the various changes in the colour of the mucous membrane, the source and character of the discharges, the appearances of partial hypertrophies of the labia uteri, and of ulcerated surfaces, may be ascertained. Sometimes, indeed, the sense of sight, aided by the speculum, may discover abrasions and superficial ulcerations, which practised touch fails to detect; and it is well known that soft mucous polypi of small size sometimes associated with abrasions, and attended by hæmorrhage and leucorrhœa, often elude the sense of touch but are readily recognised on visual examination.

By the uterine sound the practitioner is further enabled to ascertain when hypertrophy has taken place either in the cervix or body; the thickness of the uterine walls; the direction and length of the uterine cavity; the mobility of the uterus as indicating whether it has become fixed by inflammatory adhesions or not; the degree of sensibility, or the reverse, in the mucous membrane lining the uterine cavity; the presence of stricture in the cervical canal or the opposite condition of undue patency. The elongation of the uterine cavity has been regarded as affording evidence that the body of the uterus is the seat of hypertrophy or inflammation, but other affections of the uterus are also attended by increase of measurement of the cavity. This is the case when the cervix is elongated, when there is sub-involution, and in some of the forms of fibroid tumour. Not infrequently also the fundus uteri is pulled upwards, and its cavity extended by the growth of an adherent ovarian tumour.

In endo-metritis the uterine cavity has an increased sensibility. The passage of the sound produces great pain, and is often followed by bleeding and persistent uneasiness for some time afterwards. In many women who are not the subjects of uterine inflammation, the passage of the sound gives pain, particularly when the fundus is reached, but when endo-metritis is present the suffering produced is quite beyond the measure of what occurs under like circumstances in the healthy womb, although the sound may have been passed with great facility on account of the dilatation of the os uteri internum.

In reference to the employment of these instruments, it may be remarked that in most cases they are useful aids rather than essentials to correct diagnosis, and that if the sense of touch be carefully cultivated, they will, in multiple instances, but confirm what has already been made out by digital examination. The use of the speculum for diagnosis in *all* cases of chronic inflammation or ulceration of the cervix is manifestly superfluous, and, in unmarried women, is commonly both unnecessary and injurious. Again, the use of the sound should be carefully avoided whenever increased sensibility seems to indicate inflammatory action still in progress, as by its injudicious employment a further impetus may be given to inflammatory progress.

DIAGNOSIS.—An attack of acute metritis has some symptoms in common with abdominal peritonitis, with pelvic peritonitis and cellulitis, and with ovaritis. In abdominal peritonitis the pain and tenderness are more general, while, with the localisation of the suffering in metritis, there are probably some concurrent symptoms in the history of the attack pointing to the uterus as the organ attacked. In acute ovarian inflammation, the pain is in the iliac regions of the pelvis rather than in its centre, and if a vaginal examination be made, the affected ovary will probably be found swollen and tender, lying behind the broad uterine ligament or in the peritoneal *cul-de-sac*. When cellular inflammation or pelvic peritonitis is in progress, lymphy exudation speedily fixes the uterus so as to interfere with its mobility, but inasmuch as inflammation, beginning in the uterus or ovaries, often quickly extends itself to the cellular tissue and peritoneum in its vicinity, it is not easy to determine, by examination, where the action first began. The clearing up of this point is of the less consequence inasmuch as the treatment is the same in both cases. Acute cystitis may be distinguished from metritis by noticing that the urgent symptoms are centred in the bladder, while on examination, unless the attack is an extension of metritis, the uterus is found to be little affected.

Concerning the diagnosis of the results of chronic congestion and inflammation, it may be stated that, perhaps, the most frequent difficulty arises in estimating the distinctions between some of the forms of hypertrophy and ulceration produced by inflammation, and incipient cancer of the uterus. In the later stages of malignant disease the rough and irregular excavation of the ulcerated surface bounded by the sharp hardened edges, the characteristic odour of the discharges, and above all the fixing of the uterus by surrounding infiltration, render the nature of the affection but too obvious. But in the earlier stages of cancer, when the uterus is still perfectly movable, and only slight changes have occurred in the tissues of the cervix, the determination of its true character is by no means easy, and it must be in the recollection of all practitioners of experience that cases occasionally present themselves concerning which they have entertained the gravest suspicions, and which, nevertheless, have yielded to appropriate treatment. Whenever uncertainty exists it is well to give the patient the benefit of the doubt, so that the necessary methods of cure for the simpler affection, if such it turn out to be, are not omitted. It may facilitate the formation of a correct opinion to bear in mind the distinction pointed out by Dr. Bennet between simple hypertrophy of the cervix and early cancer. In the former the fissures radiate from the os uteri as a centre, which they do not do in carcinoma; and further, that there is little variation except for the worse in the malignant disease, while in the more benign affection often marked amelioration is observed for a time as the result of treatment, even in cases where recovery is slow or cure imperfect. Dr. Spiegelberg has remarked that the use of a sponge tent leaves the tissue hard and tense in scirrhous cancer, while it softens it in the benign affection. It is necessary to recollect that during the progress of metritis, if complicated with peritonitis or cellulitis, the uterus may become fixed to surrounding parts, and that the imperfect mobility of the uterus does not therefore always imply the presence of malignant disease. The enlargements of the body of the uterus from pregnancy and fibroid tumour are to be distinguished from hypertrophies the result of congestion and inflammation, by noting the associated signs of pregnancy in the first case, and the absence of inflammatory history in the last. When there is no distinct history of precursory inflammation, the diagnosis of uterine enlargement resulting from metritic changes, and fibroid tumour in the walls of the womb, is often impossible during life, and it is probable that in most instances of reputed cure or diminution in size of fibroids by Kreuznach waters and other absorbent remedies, the swelling has been due in part or entirely to hyperæmia or to unorganized adventitious deposits, capable of absorption.

The local hypertrophies which project from the posterior or anterior walls, may be distinguished from retroversion and retroflexion, from anteversion and anteflexion, by finding that the swelling can be made to disappear on pressure upwards with the finger, and if further evidence is required (as, for example, when the uterus is fixed), by ascertaining the direction of the uterine cavity with the sound.

The character of tumours which are sometimes formed in the vagina either by regular or nodular hypertrophies of the cervix, may be determined by the position of the os uteri, and by the length of the uterine cavity. In those rarer forms of tumour in which the structure consists largely of cystic growth, the distended cysts may be felt and seen scattered over the surface or imbedded in the mass, with intermediate distended veins, and the presence of the cysts is *prima facie* evidence against its malignant character.

The forms of inflammatory ulceration may be distinguished from the "*corroding ulcer*" of Sir Charles Clarke by the more rapid extension of the corroding ulcer, the alarming hæmorrhages, fœtid discharges, and indications of malignant cachexia. From syphilitic ulceration of the cervix there seems no way of distinguishing the non-specific form, except by the general history in conjunction with concurrent symptoms.

When the presence of erosion or ulceration is discovered, it is important to ascertain not only its character and extent, but also to recollect that it often forms part of a deeper-seated, and perhaps more intractable, uterine or ovarian ailment. It is essential, therefore, to proper diagnosis, not to overlook any enlargement or displacement with which the ulceration may be associated, or perchance some affection of the ovaries upon which its presence mainly depends.

To enter minutely into the differential diagnosis between chronic metritis with affections of the bladder, rectum, and other neighbouring organs, is beyond the limits of the present paper.

PROGNOSIS AND TERMINATIONS.—An attack of acute metritis either in the puerperal or unimpregnated condition is always formidable, and in the severer cases the prognosis is a grave one, inasmuch as if the patient escapes the immediate perils to life, the after results of exudation and adhesion often lead to very serious inconveniences of a chronic character. In the chronic form the prognosis is usually favourable, and cure under a suitable plan of treatment may in most cases be predicted.

Recovery from many of the results of uterine congestion and inflammation is, however, commonly tedious, and the patience of both medical practitioner and patient is often severely tried. From the anatomical position of the uterus, repose in the horizontal posture becomes almost an essential for cure, and as patients frequently object to the necessary restrictions, the progress of the affection is often unduly prolonged. On the other hand, patients willing to submit to the needful discipline for the uterine ailment, often suffer in general health from the lack of exercise and exposure to fresh air, and thus the constitutional cause out of which the local ailment may have sprung originally, is intensified. A just balance, nevertheless, may in most cases be struck between the necessities of each case, and restoration be anticipated.

In some obstinate cases the pathological condition may remain much in the same state for a prolonged period, growing neither better nor worse, even with careful treatment. The fear is that by persistence, induration and hypertrophy with their several complications, which promised at first to be only temporary, should become permanent and entail all the inconvenience of such conditions for a long future, or until the "change of life," lessens the vitality of the generative organs. It is probable also that the development of fibroid uterine tumours take their initiative in a perversion of growth arising out of persistent chronic inflammatory deposits; and there is some reason for believing that continued irritation and enlargement of the ovaries, the thickening of their coverings and the adhesions to surrounding parts so often associated with uterine inflammation, may so interfere with the performance of ovulation as to prove an active incentive to the development to cystic ovarian disease. There would seem to be little tendency for chronic hypertrophy and ulceration to degenerate into cancerous disease, but these benign alterations in structure may, in those predisposed, form a nidus for the development of malignant growth, and, by the irritation they produce in the womb, be indirectly conducive to it. Chronic enlargement of the uterus, whatever may be its cause, is a fertile source of displacement. Whenever the two conditions are found in conjunction—it matters not which has been the primary affection—the recovery is always more difficult and protracted.

Stricture of the cervical canal may likewise result from the healing of the several forms of ulceration of the os uteri, and from the effects of escharotics employed in their treatment. In this way obstructive mechanical dysmenorrhœa may be provoked after the inflammatory attack has been subdued, and the labia uteri may become so rigid as to oppose serious resistance to dilatation in a subsequent labour. Although conception may take place while some of the forms of chronic uterine inflammation are present, sterility is more frequently observed to be the rule, until the womb has returned to its normal condition again.

Treatment.—The treatment of inflammation of the womb is to be conducted on the same principles as those applicable to inflammation of other internal organs, special regard being given to its position in the economy and the peculiarity of its functions. In the acute form all examinations should be made as seldom and with as much gentleness as possible. No instruments should be used in diagnosis, and no irritating or astringent local applications should be employed in the treatment. Venesection may be practised in some cases, where the patient is robust enough to bear it and the attack is urgent enough to call for it. Local depletion is desirable in nearly all cases, and as the probable tenderness of the genital canals precludes the introduction of a speculum, leeches may be applied either to the hypogastrium or round the anus as more directly abstracting blood from the pelvic blood-vessels. The earlier the bleeding is had recourse to, the more obvious generally is the result, and the more perfect the relief. The bleeding may be repeated according to circumstances. Opium will be required in some form in frequently repeated doses, and it may be conveniently combined with tartarized antimony to lessen the force of the circulation and promote diaphoresis in the earlier stages; and with calomel and perchloride of mercury in the latter periods, to promote absorption of exudation. Dr. West speaks highly of the extract of belladonna as an anodyne in these cases, and he prescribes it in doses of one-sixth to one-quarter of a grain every four hours, increasing the quantity if necessary. Belladonna may also be usefully employed externally during the decline of acute metritis. When the need for poultices and fomentations is past, equal parts of ungt. belladonn. and ungt. hydrarg., may be spread over the surface of the abdomen and covered with flannel. These remedies may be supplemented by salines such as the acetate of ammonia or citrate of potash, when there is much febrile disturbance, and more especially when the urine is scanty and loaded with lithates. If the patient is able to leave her bed, a warm hip-bath may be permitted, and the continuous use of hot linseed poultices with laudanum or tincture of belladonna over the surface rarely fails to afford relief to the severe pain. If required, counter-irritation may be produced by sinapism or turpentine over the abdomen, but blistering by cantharides should be avoided, lest it aggravate dysuria. If the bladder is much disturbed, its irritability may be soothed by drinking freely of demulcent fluids, such as barley-water and linseed-tea, and if there is excoriation of the vagina from irritating discharges these fluids may be injected. It is well not to disturb the patient by aperients at the first outset of the attack, but no long time should be allowed to elapse without relieving the bowels by a gentle laxative or an emollient enema. If the bowels are irritable and diarrhœa occurs, Dover's powder may be given with hydrarg. c. cretâ., or an opiate enema or suppository may be prescribed for the double purpose of allaying irritation of the bowels and quelling the paroxysms of uterine pain.

The patient should maintain absolutely the recumbent posture, and have the pelvis somewhat elevated, by way of obviating as much as possible the effects of gravity. The diet should be simple and unstimulating, no wine or spirits being permitted until the acute symptoms have subsided, unless there be manifest failure of power and stimulants become essential. The treatment of the after results is essentially the same as that applicable to the various forms of chronic inflammation, into which the more acute form is apt to merge when imperfectly cured, and great care should be taken when the patient is apparently well to guard against the danger of relapse for a considerable period. More especially ought there to be absolute repose at the menstrual periods for two or three months after.

The treatment of uterine congestion and of chronic metritis must necessarily be modified by the form the malady assumes, by its locality, by its duration, by the extent and gravity of the symptoms with which they are accompanied, and by a diversity of other matters requiring careful consideration.

Not the least important is the prophylactic or preventive treatment. In all cases where either the antecedent history, or where conditions are known to exist which may favour internal congestion or inflammation, special precautions should be taken. When it has been ascertained, for instance, that sub-involution after delivery is present; when there is fibroid tumour or displacement of the uterus; much rest in the recumbent posture should be enjoined at all times, but especially at the catamenial periods. The same precaution should be observed after abortion; when ovarian irritation or over-frequent menstruation is present; and in cases of habitual dysmenorrhœa. The importance of rest, within such limits as the general health will bear, so as to obviate the effects of gravity and locomotion, can scarcely be over-estimated, and it must be within the experience of most hospital physicians how constantly all measures of treatment without rest fail to prevent and cure uterine congestion and inflammation, and how frequently repose with but little other treatment suffices for the recovery of the patient.

As in a considerable number of cases, congestion and chronic metritis take their origin in faults in general health, or, being incidentally produced by other causes, are prolonged and intensified by constitutional disturbance, great care should be taken from the outset to trace out any existing systemic derangements or any disturbance in other and distant organs which may possibly be overlooked in the prominence of the uterine symptoms. The late Dr. Edward Rigby especially insisted upon the dependence of chronic uterine ailments on constitutional causes, and urged the importance of rectifying faults in the general health as a preliminary to all other treatment. More particularly he dwelt on the necessity of carefully regulating the functions of the chylopoietic viscera, as obstructions in the portal circulation greatly favour congestion of the pelvic blood-vessels. As a first and most important preliminary, therefore, the condition of the general health should be carefully inquired into, and its faults corrected if need be. If there be evidence of plethora on the one hand, or anæmia and asthenia on the other, these should be combated by appropriate means. Injurious or enfeebling habits of life should be rectified,—any depressing influences, such as over-lactation, habitual discharges, loss of blood from hæmorrhoids, sources of mental anxiety, &c., should be removed if possible. Good food, warm but light clothing, fresh air, with exercise appropriate to each case; and mineral acids, with iron, or vegetable tonics, should be prescribed as required; the bowels should be carefully regulated, and if the liver is habitually sluggish, some suitable alterative may be prescribed from time to time. There should be abstinence from marital intercourse; violent or excessive exercise, such as long walks or dancing, should be prohibited, and it may be necessary to discontinue both riding on horseback and carriage driving, even when moderate walking is permissible. There should be no undue pressure upon the pelvic organs by the abdominal viscera, and faults in dress conducing to this should be corrected. It may be desirable indeed to raise up the abdominal organs by a bandage when too much laden with fat, or when the abdominal walls are much relaxed. When any particular diathesis is present it commonly gives a type to the local ailment, even if not directly concerned in its production. The painful affection described by Gooch as "irritable uterus," which is closely akin to an inflammatory condition, is often distinctly associated with a constitutional tendency to rheumatism or neuralgia, and is only to be relieved by prescribing for the prevailing diathesis. Other forms of chronic uterine inflammation seem to depend on a gouty constitution, and require special treatment in accordance with the pathology of that condition. When organic disease exists in the uterus all possible precautions must be taken to lessen the accidental sources of irritation associated with its presence there. Successful treatment further requires the recognition of the inter-dependence of some pathological conditions on others, and the necessity of treating more especially the affection which may be the keystone to the rest. Thus continued endo-metritis or cervicitis may produce both tumefaction of the uterine walls and erosion or ulceration of the os uteri; then treatment must mainly be directed to cure the disease in the uterine cavity. Mucous polypi, hidden in the cervical cavity, will produce hyperæmia, and persistent hyperæmia of the uterus generally will not only originate and perpetuate enlargement of the uterus, but lead to cervical catarrh with abrasion and ulceration of the cervix. When such is the order of pathological events, it is obvious that mere alterative and astringent applications to the surface inflammation about the os tincæ, will have little curative effect unless measures be taken to remove the exciting cause.

If the uterus is in any way displaced, the necessity of readjusting its position must be kept in view as soon as this is practicable.

The theory promulgated by M. Velpeau, and subsequently resuscitated by Dr. Graily Hewitt, that patients suffering from sympto of uterine inflammation are almost universally found to be affect with flexion or version of the organ, and that the treatment f displacement is therefore, as a rule, of primary importance in the cases, is not in consonance with general experience. It is importa however, to recognise the fact that displacement may in certa patients, originate and perpetuate uterine inflammation until it rectified, and that flexion and version may take place as seconda complications of congestion and inflammation, when the fundus h grown heavy, its ligaments relaxed, and the tissues of the womb have become softer than usual. The employment of mechanical appliances, even when these are thought necessary, is certainly counter-indicated when there is any undue tenderness about the uterus and its appendages, and under these circumstances it is best to postpone the rectification of position by mechanical means until pain and sensitiveness have abated. The postural methods for displacement may without fear be recommended, as they necessitate no manipulations of the tender womb. When the sensitiveness has passed, some form of vaginal pessary, suitable to the circumstances—or a pelvic belt—may be employed, and if carefully adapted, the support will then give much relief, even when enlargement proves too obstinate to be removed by the remedial measures prescribed. In habitual prolapsus, the elevation of the uterus and the retention in its natural position is almost essential to the prevention and cure of the hyperæmia and other morbid results which ensue from the interrupted circulation and exposure to mechanical injury.

The local treatment of congestion, induration, thickening, and hypertrophy of the body and cervix, consists therefore, in the first place, in the enforcement of such rest in the recumbent posture as the constitutional condition will bear. Next, the local abstraction of blood by leeches or scarification is of great value. On the continent, the derivative effects of repeated small bleedings from the arm has been lauded as offering greater advantages in promoting cure than local depletion, but in Great Britain this method is little practised. In this country leeches are commonly applied when deemed necessary either to the hypogastrium, to the anus, or to the cervix uteri. When the capacity of the vagina and the absence of tenderness will permit, leeches are best applied to the cervix by means of a small speculum or leeching-tube, for in this way the most decided results are produced upon the over-distended blood-vessels. When possible, it is best to choose a time for the abstraction of blood midway in the menstrual interval, so as to disturb as little as possible the regular return of the catamenia. Where, however, the flow at the period has been very scanty, attended with much pain, and it is desired to supplement the sparing discharge, the leeches may be employed immediately after. If scarification be preferred, it can be performed through a speculum by an appropriate lancet with a sufficiently long handle. To procure a free bleeding, the scarifications must be numerous; and punctures an eighth of an inch deep into the uterine tissue produce a more effectual flow than mere surface scarification. Neither scarification nor puncture as a rule give much pain to the patient, and when leeches are not available they offer a ready substitute, but often seem less effective. The quantity of blood to be abstracted must necessarily vary with the result to be attained and the strength of the patient. With symptoms very active, it may be necessary to apply from three to six leeches and repeat them after an interval of three or four days; in other circumstances a small depletion once in the menstrual interval and repeated a month later may be all that is required. The flow of blood is favoured by the use of fomentations and warm hip-baths. Blood may also be abstracted by cupping over the sacrum. The results to be attained by blood-letting may be further advanced by counter-irritation. Dry cups and vesicants may be used over the sacrum, or the skin may be irritated by croton oil and tartarized antimony. If the surface of the abdomen is selected for counter-irritation it is well to avoid the use of cantharides lest it provoke strangury, but strong tincture of iodine or successive sinapisms may be advantageously employed in this locality. The systematic use of a warm hip-bath is a further valuable adjunct to other treatment, and when medicated either by some sedative or absorbent, possesses additional advantages. The iodo-bromated waters of Kreuznach, Hale, Durkheim, and Krankenheil, have a great reputation for reducing adventitious enlargements of the uterus in chronic cases, and Woodhall Spa in this country is reported to have similar properties. An artificial Kreuznach bath may be extemporized by adding half a pound of Kreuznach salt and two pounds of common salt to three gallons of warm water in a hip-bath. The patient remains in this from fifteen to thirty minutes, and it may be used every night or every second night. At Kreuznach, it is customary to apply the concentrated water or "mother lye" in the form of compresses across the abdomen, and advantages may also be obtained by the frequent use of a continuous tepid douche, to the water of which is added either a proportion of Kreuznach water, or Kreuznach salt, or, when these cannot be obtained, small quantities of tincture of iodine or iodide of potassium. If there be much local pain, it may be desirable to recommend the injection of warm poppy decoction into the vagina, or a lotion composed of warm Goulard water and laudanum, in the proportion of a pint of the former to one or two drachms of the latter. The medicated pessary also offers another mode of applying remedies to the uterus, and in this form belladonna, atropine, morphia, and other sedatives may be introduced into the vagina. Bromide of potassium, iodide of lead, and other remedies may be used as absorbents in the form of pessary, either alone or in combination with sedatives. It must be recollected, however, that double the quantity of morphia, or other sedative administered by the rectum, requires to be used by the vagina, as the genital canal has a feebler absorbent power. Formulæ for the different varieties of medicated pessary, will be found in the supplement to Squire's Companion to the British Pharmacopœia. The internal remedies found useful in reducing the size of the congested and inflamed uterus are the various salts of bromine and iodine, and alterative doses of mercurial preparations. Dr. Oldham, some years ago, recommended small doses of perchloride of mercury for reducing the enlargement of a uterus produced by chronic inflammation, and there is perhaps no remedy more potent in suitable cases. Given with bark and other tonics, it may be administered for prolonged periods, and commonly without constitutional disturbance or ptyalism.

In protracted cases of inflammatory thickening of the cervix, advantage may be obtained by repeatedly painting the surface, through a speculum, with tincture of iodine the strength of the Edin. Pharm., on the same principle as iodine is applied to enlarged glands and tonsils; or a pledget of iodized cotton may be applied, as recommended by Dr. Greenhalgh. The iodine seems to have the double effect of an absorbent and counter-irritant when so applied. Repeated applications to the cervix of a tampon soaked in a mixture of one part of tincture of iodine and two of glycerine, and subsequently covered over with cotton-wool, have proved useful in these conditions. Glycerine alone even seems to act as a local hydragogue and to promote subsidence of the swollen tissues. Should more energetic measures be thought necessary, internal counter-irritation may be produced by rubbing solid nitrate of silver over the cervix uteri until a breach of surface is made over the mucous membrane, or blisters may be produced by carefully painting vesicating collodion or *acetum cantharidis* on the uterine neck, through a speculum, as recommended by M. Aran. A flow of serum is the result, which is said to act as a useful derivative. Great caution is required in following out this practice. When such hypertrophy of the cervix exists that it simulates prolapsus, and resisting all treatment seriously interferes with the comfort of the patient, it has been proposed to remove the portion of the cervix below the reflexion of the vagina by amputation. This operation may be successfully performed with the *écraseur*—great care being taken so to limit the part removed that the peritoneum behind is not endangered; or the part may be removed as recommended by Dr. Marion Sims by first dissecting back the mucous membrane of the cervix, removing the hypertrophied structures, and afterwards bringing the dissected parts together like the flaps of an amputated limb.

In the treatment of chronic inflammation of the mucous lining of the uterus the same general rules must be observed as those laid down in the last section, and this more especially in view of the fact previously pointed out that inflammation of the mucous membrane rarely persists for any length of time without extending to the middle coats and producing characteristic changes there as well as in the ovaries. In both the *membranous* and *catarrhal* varieties much rest is essential, and special precautions should be observed at the catamenial periods. Sexual intercourse, if not entirely prohibited, should be at distant intervals. Where there is evident turgescence of the uterine tissue appreciable to the sense of touch, or a congested appearance of the mucous membrane of the cervix, as seen through a speculum, and particularly if the parts are tender to pressure, the application of leeches is indicated between the periods. A course of perchloride of mercury with tincture of bark, followed by bromide or iodide of potassium, or both combined when the patient's strength will permit, often renders signal service. Unfortunately these remedies are not well borne when there is anæmia and much general feebleness. In these cases the iodide of potassium may speedily produce the ordinary symptoms of iodism, and although the bromide may occasionally be taken for months without impairing nutrition, yet women often complain, while under its influence, of irritation produced on the skin, and the constant recurrence of crops of acne. This may oblige its use to be relinquished.

The late Dr. Dewees of Philadelphia recommended the administration of the ammoniated tincture of guaiacum, in the membranous form of dysmenorrhœa, and stated that it had proved remarkably efficacious as a method of relief and cure in his hands. It is probably best adapted to those cases of dysmenorrhœa which are associated with a rheumatic diathesis, but as an alterative it has proved useful in other forms of chronic uterine inflammation, promoting activity in the various secretory organs, and acting as a laxative on the bowels. The pain experienced at the monthly periods may be palliated by the use of warm hip-baths and fomentations in those cases where the loss of blood is not excessive, or likely to be unduly increased by such measures. Further relief may be afforded by opium or morphia, administered either by the mouth, rectum, or by hypodermic injection. As the pain is frequently more or less spasmodic in character, sulphuric ether or valerian may be combined with laudanum or compound tincture of camphor. If the use of opium or morphia is precluded on account of the sickness or other discomfort it produces, it may be replaced by suitable doses of iodoform, Indian hemp, henbane, conium, or belladonna. A useful formula for relieving suffering at the menstrual periods in these cases without having recourse to opium, consists of a third of a grain of extract of belladonna, three grains of camphor, and enough extract of henbane to make a pill. This may be given every three or four hours. *Apiol*, a volatile oil from the parsley tribe, is given in France for the same purpose. The dose is inclosed in a gelatine capsule by way of avoiding its pungent taste, and in the experience of the writer it has often given notable relief, but whether it possesses any other property than that of a warm diffusible stimulant is not well determined. As a radical method of cure in cases of membranous inflammation, it has been proposed to cauterize the interior of the uterus by nitrate of silver or other caustic. There seems no evidence as yet, however, of the utility of this practice, and when it is recollected that this form of uterine inflammation is commonly associated with, perhaps essentially dependent on, ovarian irritation, local applications to the interior of the uterus do not afford much promise of ultimate benefit, and it is best to rely mainly on the general remedial measures employed in chronic congestion and inflammation of the uterus.

In treating cases of chronic uterine catarrh, or in other words the catarrhal variety of chronic endo-metritis, it is necessary to recognise two forms which require different methods of manage-

ment. Thus, wherever, in conjunction with habitual hy tion
from the uterine cavity, there is pain and tenderne
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possibly undue throbbing of the uterine arteries, wit
mixture of blood in the discharges, the most appropria
is absolute rest, leeching, the use of local sedatives a
injections, and the administration of such remedies as were
mended for inflammation of the proper substance of the
Under these circumstances all local treatment by strong astringe
or caustic applications is contra-indicated. It is commonly found, indeed, that the sudden checking of secretion provokes more suffering, and probably produces more profound lesion.

When the symptoms indicative of more active mischief are absent, or have been subdued, the necessary measures for improving the condition of the mucous membrane, and thus arresting the undue secretion from its surface, may with propriety be undertaken. In this phase uterine catarrh is very generally admitted to be in most cases a purely local affection, and to be properly treated by local remedies. At the same time to effect a cure by any process is avowedly difficult, and so eminent an authority as Scanzoni does not remember that he has been fortunate enough to cure any instance of several years' standing. Recent improvements, however, in the way of medication and local treatment afford much greater promise of affecting cure than formerly, particularly where the disease is confined to the canal of the cervix. The less obstinate forms of the affection are indeed sometimes efficiently treated by the use of astringent vaginal injections and medicated pessaries. By way of saving the patient the inconvenience of direct applications, necessitating frequent vaginal examinations, such methods as can be carried out by the patient herself may first be tried. Thus, injections of the salts of zinc and alum, and carbolic acid sufficiently diluted, of tannin and perchloride of iron, thrown against the os uteri by an efficient douche, may be employed. The iron alum recommended by Dr. Tyler-Smith in his work on leucorrhœa, is a very valuable astringent for this purpose. Independent, however, of the effect of astringents dissolved in the injection, much good is sometimes attained by the diligent use of plain water in a vaginal douche; and a stream of cold or tepid water made to play forcibly against the cervix uteri night and morning for a considerable period, often gives fresh tone to the parts, and promotes ultimate cure. Tannin, gallic acid, common and iron alums, carbolic acid, persulphate and perchloride of iron, may also be used by the patient herself in the form of pessary, the horizontal posture being maintained during their solution, and the pelvis being elevated to ensure the flow upwards of the melting material. Probably the most powerful astringents used in this form are the perchloride of iron and the iron alum.

In more obstinate and intractable cases further treatment becomes absolutely necessary, and various methods have been proposed for applying remedies more certainly to the interior of the uterus. Thus, solutions of nitrate of silver, sulphate of zinc, chromic acid, tincture of iodine and acid nitrate of mercury, have been injected into the uterine cavity by means of a properly curved silver catheter with an attached syringe. Unfortunately all such irritating injections are apt to produce severe uterine colic, with grave nervous disturbance, unless the quantity of fluid injected be very small, or unless the os uteri be so patent as to permit the speedy discharge of the liquid. Both acute metritis and peritonitis, and in very sensitive patients alarming convulsions, have followed such proceedings. To obviate this inconvenience it has been proposed first to dilate the os uteri with a sea-tangle or sponge-tent, and inject immediately afterwards. The tangle-tent in dilating often provokes more severe pain than the sponge-tent, particularly if there be any rigidity of tissue, so that the latter should be preferred. Dr. Emmet of New York maintains that the elastic dilating pressure of sponge-tents of itself is an efficient means of diminishing hypertrophied conditions of the cervix and of curing granular erosions there. Be this as it may, the use of a sponge-tent has, besides opening up the os uteri to permit the escape of injected fluid, the obvious advantage of expanding the deep folds of mucous membrane lining the cervix and thus exposing the surface more fully to the agent employed. It is thus an especially important aid in treating chronic inflammation of the cervical canal. The crypts here are ordinarily so clogged with viscid mucus that to ensure the same end M. Huguier recommends scarification of the interior of the cervix before cauterization there. After dilatation and the careful removal of mucus by small pieces of sponge or suction with a syringe, one or two drachms of tincture of iodine, solution of nitrate of silver (grs. xx. ad ℥j), or solution of perchloride of iron with glycerine, may be injected, and repeated according to circumstances, a fresh dilatation with sponge-tents being made on each occasion.[1] When using strong injections it is desirable to use a glass speculum, so that the fluid in returning shall flow as little as possible over the vagina, and it is especially important not to throw an injection into the upper part of the cavity of a flexed uterus, as the fluid may be retained above the bend and entail all the evils of a contracted os uteri. Some practitioners prefer the application of solid substances to the interior of the uterus to intra-uterine injections, and for this purpose various medicated astringent bougies and porte-caustiques have been invented. M. Huguier applied solid nitrate of silver after superficial incision before mentioned. Sir James Simpson was in the habit of introducing a piece of solid lunar caustic into the uterine cavity, which in dissolving spread itself over the mucous membrane. M. Courty recommends the same method, and speaks highly of its efficiency, while he has not met with any untoward results from the practice. Dr. Athill and others strongly advise the application of fuming nitric acid to the cavity of the uterus after dilatation and other suitable precautions, and urge that the practice is neither hurtful nor painful. Professor Thomas of New York advocates the use of medicated tents, containing iodine, chromic acid, &c. These are passed into the uterus once a week and allowed to remain for some time, so that the medication may come thoroughly in contact with the whole surface. A plan recently recommended by Dr. W. S. Playfair is to carry concentrated carbolic acid by means of a flexible metallic probe armed with a thin covering of cotton-wool as far into the uterus as may be possible. A patulous condition of the orifice favours its introduction. The pure carbolic acid rendered fluid by the addition of 20 per cent. of water may be used in this way from time to time without causing much pain. It possesses advantages over both nitrate of silver and acid nitrate of mercury employed by some practitioners, and undoubtedly answers very well in many cases. To get the full effect, the tough mucus must first be carefully removed from the mucous membrane by bits of sponge or cotton-wool on a probe before applying the acid. The perchloride of iron dissolved in glycerine (ʒj ad ℥j), may be applied in the same way. This agent has the peculiar property of so thoroughly coagulating the viscid mucus lying in the cervical canal, that it forms into dry crumbs, and thus enables the canal to be thoroughly cleared before using a second probe medicated either with iron perchloride or with carbolic acid. Pledgets of cotton-wool furnished with a string to facilitate their removal and soaked in tannin and glycerine, or perchloride of iron and glycerine, may be applied to the os uteri twice or thrice a week, and sometimes answer very well. Even plain glycerine used in this way promotes cure, in virtue of its penetrating power and capability of attracting other fluids. Dr. Braxton Hicks uses pencils of sulphate of copper, iron, zinc, and alum, which have been cast in a thin mould the size of the cervical canal; and Dr. Matthews Duncan speaks highly of the utility of zinc alum, a compound of zinc and alum similarly prepared. These methods of treatment are especially applicable to the forms of endo-metritis in which the disease is chiefly confined to the cavity of the cervix. In certain cases where the effect of chronic metritis has been to produce a permanent alteration of the glandular and villous structures, and a thickened and fungous condition of the lining membrane attended by menorrhagia or the frequent admixture of blood with leucorrhœal discharge, Sir James Simpson, Dr. Routh, and others have suggested that the cavity should be scraped by a curette with a cutting edge, somewhat similar to the curette of Recamier, and Dr. Routh afterwards applies tincture of perchloride of iron, iodine, or other substances to the abraded surface, by way of inducing more healthy action. Incision of the cervix has also been recommended as preliminary to other treatments, and is indicated if the external os is much contracted. It may be observed, however, that the greater the interference by such violent methods, and the stronger the local application, so is the risk of lighting up fresh complications. These forms of treatment, therefore, should be reserved for exceptionally obstinate cases. There are few instances which do not ultimately yield to some of the less violent methods previously described if the general health be at the same time improved. The waters of Ems, Selters, and Luhatschowitz are held in much esteem on the Continent, for their efficacy in uterine as well as other forms of catarrh. Of these the springs of Ems enjoy the greatest reputation, and although the waters have been probably overrated as remedies for all the forms of chronic metritis, there can be no doubt of their utility in cases of chronic uterine catarrh. The thermal vaginal douche of the Bubenquelle spring is a very powerful remedy in this affection, but its use needs carefully regulating, as incautiously employed it often gives rise to considerable pain, and indeed the too prolonged or unguarded use of the Ems waters in any way frequently gives rise to much constitutional feebleness and relaxation. To remedy any debilitating effects so produced it is commonly recommended that patients shall take a supplementary course of chalybeate waters at Schwalbach, Spa, or Pyrmont, or combine some Schwalbach waters with the waters of Ems while pursuing the course at that place.

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[1] Mr. Coxeter, of Grafton Street, and other instrument makers, sell small syringes specially adapted for uterine injections.

Concerning the treatment of abrasions, erosions, and ulcerations of the os and cervix uteri, much controversy has taken place, and the questions as to whether local treatment is required, and as to what extent, and kind, have been hotly contested. There would seem to be some ground for believing that local methods of cure have been carried to an extreme by some practitioners who take too limited a view of the nature of these alterations, and do not appreciate their true pathological import. Dr. Edward Rigby, indeed, did not scruple to aver that certain medical men had behaved dishonestly in having recourse to local applications when uncalled for, and hinted that cauterization had, within his experience, been practised simply to impress the mind of the patient.

Viewing these deviations from healthy conditions as strictly analogous to pathological changes of like character occurring in other parts of the body (and from this aspect alone can any rational principles be laid down for their treatment), it is obvious that the same form of procedure is not applicable in all cases, but that variations of condition require modifications of treatment. It would be the merest empiricism to cauterize all forms of surgical ulcer, and equally it is an excess of zeal, often most injurious in its effects, to treat all erosions and ulcerations of the os uteri in this way. Recollecting that a large proportion of these abrasions and erosions do not reach the stage of true ulceration, and often bear a resemblance both in appearance and pathological import to the granular conjunctiva, or it may be to a crop of herpes on the mouth, it may clearly be inferred in these cases that little local treatment is required. Practically it is found that cleanliness, with rest and careful attention to the constitutional condition which may have been the cause of the local ailment, is sufficient for the cure. When further treatment is required, and there are evidences of local relaxation, the cold or tepid alum hip-bath, consisting of half-a-pound of powdered alum dissolved in three gallons of water, proves a useful adjunct, and, in the case of mc rried women suffering from the simple forms of leucorrhœa, the administration of a ferruginous tonic, or of a mineral acid table tonic, and a nutritious diet, is all the treatment requ aid may be derived in suitable cases from the use of zi solutions by vaginal injection. The cold or tepid vagi o efficiently managed is a potent remedy for imparting fresh tone to parts. A Kennedy's or Higginson's syringe may be used for this p pose, or the douche recommended by Dr. Graily Hewitt in his wo on the diseases of women. The materials found most useful for medicating vaginal injections are the common and iron alums, chloralum, sulphate of zinc and sulpho-carbolate of zinc, tannin and gallic acid, perchloride of iron and carbolic acid; Goulard water and infusions of oak bark or matico may likewise be employed. Recently the various forms of medicated pessary previously alluded to (pp. 738) have been much used either to replace vaginal injections, or as additional aids, and when properly managed they have the obvious advantage of keeping the medication for a longer period in contact with the affected part. Further, medicated pessaries have the additional advantage of being appropriately used during pregnancy, when frequent douches and injections may not be employed without the risk of separating the membranes and provoking abortion. In a large proportion of the simpler forms of ulcerative inflammation of the cervix the treatment above described suffices for the recovery of the patient. In more obstinate cases, where, it may be, true ulceration has been produced, or where some complication exists, other measures of treatment will be required. And here it is important to recall the fact that erosions and granular ulcerations of the os uteri are often secondary to other and more important uterine affections, and that to effect a cure of the primary ailment which may have been the cause of ulceration is of the first consequence. Thus the presence of endo-metritis, of mucous polypus, of uterine displacement, or hypertrophy, should be sought for, and, if found to be factors in the case, should be appropriately treated. In all cases, the cervix should be kept as much as may be from the injurious influence of friction. Coitus should be forbidden, and if prolapsus is present, the womb should be supported by a ring horseshoe, or other pessary which cannot chafe the granular surface. Where there is habitual congestion, or, it may be, thickening and induration associated with ulceration, and particularly if at the same time there be pain and tenderness, the local abstraction of blood by leeches or scarification in accordance with the rules laid down in a previous page, will be an essential preliminary to other treatment. Instead of astringent injections and pessaries, more soothing remedies must be employed, and warm hip-baths and injections of warm poppy decoction, or of lead and opium, may be substituted. Instead of astringent pessaries those containing morphia, belladonna, and atropine may be prescribed. Occasionally mercurial ointment, bromide of potassium, iodide of lead, bismuth, and oxide of zinc, are combined with a sedative in this form. The dry powders of calomel, bismuth, and oxide of zinc have also been shed by various contrivances over the affected parts. When there is much tendency to excoriation, heat, or pruritus, borax may be combined with a sedative either in injection or pessary. Hydrocyanic acid with emulsion of bitter almonds, or hydrocyanic acid and solution of morphia, may be added to Goulard water in the proportion of two drachms of the former ingredients to eight ounces of the last. If there be offensive irritating discharges the permanganate of potash and the sulphocarbolates in lotion, give marked relief. Attention also has recently been drawn to boracic acid lint as a potent application for pruritus.

When tenderness has for the most part subsided, and the indications of active and progressive mischief in the deeper structures are in abeyance, the persistence of a granular condition or ulceration of the cervix may necessitate some form of cauterization, or the application of some astringent stronger than can safely be employed in the form of injection or pessary. The agent which has been most extensively used for this purpose is lunar caustic, and Dr. Henry Bennet and other eminent authorities speak very highly of its efficiency. The nitrate of silver is so readily decomposed by the mucous discharges, that the surface needs to be carefully cleansed by cotton-wool or bathed with water before its application. Solutions varying from twenty to sixty grains to ℥j. of distilled water are to be preferred when the granulations are very vascular, as then the use of the solid caustic often provokes bleeding. The weaker solutions are best suited to the more irritable forms of ulcer, the stronger to the more sluggish and insensible. An application of this kind once or twice a week commonly suffices. Other materials used in the same way are carbolic acid, perchloride of iron, tannin and glycerine, Richardson's colloid styptic, the sulphates and chlorides of zinc, sulphate of copper, the acid nitrate of mercury, butter of antimony, chromic acid (℥ss to water ℥j.) nitric acid, iodoform, &c. All the stronger substances require the use of a speculum so as to insure the limitation of the medicament to the part affected. Less potent and less irritating solutions may be applied by means of Sir Charles Locock's tube—a glass syringe open at its extremity and furnished with a sponge which can be projected beyond it. Carbolic acid possesses special advantages for the purpose of these applications. It does not leave indelible stains on linen like nitrate of silver, and as pointed out by Neumann of Vienna, it causes the tissues to shrink and their fluids to coagulate, while it rarely makes even the most vascular surface bleed. It may be successfully applied in its concentrated form to the exuberant development of villi, which has been called "cock's-comb" granulation, and to the sort of hæmorrhoidal condition of the os uteri, consisting of eversion of the mucous membrane, with distended capillary vessels, described by Sir J. Y. Simpson. Unlike potassa fusa and other strong caustics it does not leave an eschar, and thus is not likely to be followed by after contraction of the cervical canal. It may be applied in small quantities to the part with a brush in its concentrated form, enough water only being added to make it fluid, or a small pledget of cotton-wool soaked in the glycerinum acidi carbolici of the *Ph. Br.* may be pushed through a speculum to the affected part and there allowed to remain some hours covered by a larger one soaked in simple glycerine. When the morbid condition extends upwards in the cervical canal, the application should be made to follow it there, so as to insure a more complete healing. Its effect in promoting rapid cure is sometimes remarkable. When there is much thickening or induration in the deeper structures subtending granular erosion or ulceration, the tincture of iodine as before mentioned may be preferred to any of the more direct astringent or caustic remedies, as to promote absorption in the deposit beneath is the best way to insure the healing of the broken surface.

Whether it is desirable in cases of considerable and obstinate hypertrophy of the cervix, associated with ulceration, to have recourse to more severe forms of cauterization, is still a debated question. Sir J. Y. Simpson recommended the use of potassa fusa under the circumstances. The part being well exposed in a glass speculum, a stick of caustic potash is pressed firmly upon any thickened part, until a slough is produced; the parts being freely bathed with acetic acid immediately after to limit the effect of the escharotic. The object is to stir up healthy action in the part, and favour ultimate absorption of the exudation. Sir James asserted that in a day or two after such cauterization the slough separates and leaves a healthy sore on a softened cervix, which soon heals under the application of some astringent injections or pessaries. Dr. Henry Bennet, another eminent authority, prefers the potassa c. calce, a somewhat diluted preparation, with the same objects; and intimates that it may be necessary to make the application several times, the application being each time limited to a small space. M. Joubert de Lamballe and other practitioners in France employed the actual cautery, and the recent improvements in the forms of galvanic and gas cauteries have greatly facilitated the application of the actual cautery, and do not so readily alarm the patient. Unfortunately there are grave objections to the frequent use of these powerful escharotics, and they should consequently be reserved for exceptional instances. The contraction of the cervical canal thus produced may subsequently so interfere with the escape of the menstrual discharge as to lead to severe dysmenorrhœa. Dr. Bennet has further pointed out that the use of potassa c. calce, although it may not inflict much pain, produces great prostration of strength; and there can be no doubt that caustic potash frequently leaves among its after effects severe uterine neuralgia. The use of strong escharotics is nevertheless justifiable when partial nodular hypertrophies are present which resist milder forms of treatment, and which provoke frequent hæmorrhage; and the same may be said of the fungoid condition produced by simple hypertrophy of the papillæ and glandular follicles around the os uteri, under similar circumstances. Some of these hypertrophies only involve one labium uteri, and consequently there is no occasion to apply the cautery to the whole circle of the os uteri.

In most other cases absolute rest, local depletion, counter-irritation, small doses of perchloride of mercury, and careful attention to diet, regimen, and those minor measures found useful generally in uterine complaints, will promote the recovery of the patient with less risk and almost as certainly as the employment of strong caustics. In the rarer instances where the hypertrophy of the cervix is so considerable as to prove a serious inconvenience to the patient, the amputation of the part is rather to be recommended than the slow and uncertain action of repeated cauterization.

In the local treatment of uterine affections, it is on all accounts most desirable that vaginal examinations in married women should be reduced to a minimum consistent with the recovery of the patient. In virgins, frequent local applications by a medical practitioner are commonly both uncalled for and injurious, and it is only when all other measures have failed, and the condition of the patient urgently demands further interference, that they are justifiable.

Lastly, whenever patients, the subjects of chronic uterine inflammation, have been much enfeebled by the persistence of the malady and the treatment necessary for its subjection, some after-treatment with a view of restoring the strength of the patient becomes essential. Thus a change from town to country or to the sea-side, where bathing may be had, is followed by a marked advantage. If the patient suffers from amenorrhœa or is anæmic and feeble, the waters of Spa, Schwalbach, Pyrmont, and the mud baths of Franzenbad may be prescribed. If there be an impaired condition of the digestive organs and abdominal circulation, the spas of Vichy, Kissingen, Homburg, Carlsbad, &c., may be recommended in accordance with the special requirement of each patient. Schlangenbad and Wildbad have a large reputation for calming the nervous affections associated with uterine disorders.

BIBLIOGRAPHY.

LIST OF AUTHORS REFERRED TO.

Thomas Gaillard, Diseases of Women.
Huguier, Bull. de l'Académie de Méd. vol. xv.; Mém. de la Soc. de la Chir. vol. i.; Gaz. des Hôp. 1847.
Cruveilhier, Anatom. Patholog.
Virchow, Verhand. der Gesellschaft d. Geburt., 1847 ; Archiv. 1862.
Kiwisch, Klinik Vorträge.
Scanzoni, Diseases of Females, Amer. edit. translated by Dr. Gardiner.
Klob, Pathol. Anat. of Female Sexual Organs (Amer. edit.).
Oldham, Guy's Hosp. Reports, 2nd Series, vol. vi.
Simpson, Sir J. Y., Diseases of Women.
West, Diseases of Women, and Croonian Lect. On the Pathological Importance of Ulceration of the Os Uteri.
Rigby, On Female Diseases.
Bennet, H., Inflammation of the Uterus.
Tyler-Smith, On Leucorrhœa.
Donné, Rech. Micro. sur la Nature du Muqueuse, &c., 1837.

Snow-Beck, Med. Times, 1857.
Paget, Sir J., Surgical Pathology.
Farre, Arthur, Cyclop. of Anatom. and Physiol., article Uterus.
Robert, Des Affect. du Col d'Uterus; Thèse. Paris, 1848.
Tilt, Uterine and Ovarian Inflammation.
Matthews Duncan, Edin. Med. Journ., 1862.
Boys de Loury and Costilhes, Gaz. Méd. de Paris, 1845.
Kennedy Evory, Dub. Med. Journ., 1838.
Gooch, Diseases of Women.
Graily Hewitt, Diseases of Women.
Churchill, Diseases of Women.
Routh, Brit. Med. Journ., 1862, and Obstet. Journ., vol. iii.
Clarke, Sir Chas., Diseases of Females.
Velpeau, Bull. de l'Acad. de Méd., 1854.
Nonat, Malad. d'Uterus.
Greenhalgh, Obstet. Trans., vol. viii.
Aran, Malad. de l'Uterus, and Bull. de Thérap., 1852.
Marion Sims, Notes on Uterine Surgery.
Dewees, System of Midwifery.
Emmet, quoted by Playfair.
Courty, Malad. de l'Uterus.
Playfair, W. S., Brit. Med. Journ., 1869.
Braxton Hicks, Obstet. Trans., vol. viii.
Neumann, Wiener med. Wochenschr., 1867.

GROWTHS IN THE UTERUS.

By John Williams, M.D.

FIBROID TUMOUR.

Synonyms.—This form of new growth has received many names, as Scirrhus, Sarcoma, Fleshy tubercle, Steatoma, Sub-cartilaginous tumour, Fibroma, Myoma, Myo-fibroma, Dermoid, and Hysteroma. Fibrous or Fibroid Tumour is the term by which it is now most usually known, and serves perhaps as well as any other the purposes of designation and distinction.

History.—Though known to the ancients, yet until recent times it was confounded with growths of a much more serious character, for it was regarded as being of the nature of cancer. William Hunter first pointed out its true character, and called it "Fleshy tubercle." Yet Ashwell, more than fifty years afterwards, strongly maintained that it was a cancerous production, and gave five reasons in support of his belief, "not a single one of which is entitled to any weight." Its real structure was described by Vögel in the year 1843. He showed that it was composed of tissues similar to those of the uterus. Since that time it has been definitely ascertained that the growth, whether it occurs in the pregnant or the unimpregnated organ, possesses a structure very similar to that of the womb itself.

Pathological Anatomy.—A uterus may be the seat of a single fibroid growth, or of many. Seldom, however, is this form of tumour found solitary; several usually grow in the same organ; two or three, or as many as twenty or thirty, or more, may be present in the same uterus. It does not usually attain a great size. It varies from the size of a walnut to that of a fœtal head. Occasionally, however, it may attain very large dimensions, and cases have been met with in which the abdomen was enormously distended by the growth, and in which after removal the tumour weighed seventy or eighty pounds. On the other hand, the growth may be so small as to be overlooked even on a careful examination. I have in my possession a specimen which is not distinguishable by the naked eye, and which was accidentally found while making a microscopic examination. It is a common occurrence to find growths no larger than a pea or a bean.

Fibroid tumours, when simple, are usually spherical in shape, have a smooth surface, and are lodged in a capsule of fibro-cellular tissue, which separates them from the uterine tissue around, and out of which they can be readily enucleated. Their attachment to the uterus is by a slender cord of fibrous tissue, in which small blood-vessels run. They are hard, resisting, and creak under the knife. On section they present a somewhat undulating surface of a greyish, whitish, or reddish-white appearance, exhibiting opaque white or pearly-white striæ interlacing in various directions. Sometimes these striæ have a concentric arrangement around a central nucleus. These consist of fibrous tissue and muscular fibre cells in varying proportions, but the former always predominate. The muscular fibre cell appears to be somewhat larger than that found in the empty uterus, but smaller than that in the impregnated organ. The meshes formed by the fibrous bands contain a greyish-white substance, composed of round and elliptical cells, and these, with the fibres forming the bands, are bound together by a transparent structureless or slightly fibrillar matrix. Several such simple fibrous masses of smaller or larger size may be bound together within the same capsule, and thus form a conglomerate fibroid tumour. Such tumours differ not in structure from the simple forms just described, except that they are less dense and more vascular. The individual masses of which such compound or conglomerate growths are formed, by reason of the pressure resulting from their contact with one another, lose their spherical form, and assume various irregular shapes. They are separated from each other by processes of connective tissue proceeding from the capsule which surrounds the whole. The surfaces of such conglomerate masses are lobulated and irregular in consequence of the uneven projections of the growths entering into their formation. They possess a kind of hilus with a large venous plexus, from which veins proceed between the lobes of the tumour along the processes of connective tissue found there, receiving in their course minute venous branches from the growth. Opposite the hilus, the wall of the uterus presents a cavernous structure. While the veins are of considerable size, the arteries supplying fibroid tumours are usually minute. They enter in the band of connective tissue by which the tumour is attached to the uterine wall and ramify in the tumour. Attempts at injecting the arteries of fibroid tumours have in some instances failed. This was probably due either to some peculiarity in the growth itself, as commencing degeneration, causing occlusion of the vessels, to imperfect fluidity of the material injected, or to imperfected manipulation, for usually there is no difficulty in injecting the vessels of growths of this nature, and when injected with a solution of carmine their section presents a deep pink colour, though no vessels of any size can be distinguished in them. Though the hard fibroid tumours are usually but little vascular, it is important to bear in mind that this is subject to exception, and that occasionally an artery as large as the radial supplies the growth. The bearing of this fact upon the surgical treatment of fibroid tumours is evident.

Besides the dense, hard, whitish, little vascular growths just described, there is another variety of fibroid tumour which is less often met with, and which it is important to distinguish, for it differs from them in character, and requires a different method of treatment. This form is softer than the one described, grows to a larger size, and sometimes simulates the pregnant uterus in shape and consistence, so that some difficulty may be met with in discovering the real condition present. Barnes says,—"These tumours are not so often multiple as the hard fibroid: they almost invariably affect the body of the uterus: they attain a large size: they are softer, looser, more like muscle, have often interspaces filled with serum: they are more disposed to become 'fibrocystic:' they are not so often encapsuled. They are much less disposed to calcareous degeneration. They are more liable to become œdematous. They are more vascular, and therefore more prone under surgical interference or other violence to become inflamed, to undergo necrosis, to give origin to septicæmia and peritonitis. They are less prone to become polypoid. They frequently give rise to profuse menorrhagia."

The usual seat of fibroid tumours is the body and fundus of the uterus; it is rarely they are met with in the cervix, and still more rarely in the vaginal portion. They may, however, grow wherever tissues similar to those of the uterus occur, and they have consequently been met with in the vagina, Fallopian tubes, ligaments of the ovary, and the broad ligaments. Fibroid tumours are the most common form of uterine neoplasms. They are very common. According to Bayle, they are found in twenty per cent. of women who die after the age of thirty-five, while Klob says that they occur in forty per cent. of women who die after the fiftieth year. Dr. Charles West found fibrous tumours in the uteri of seven women out of seventy who died after the age of puberty, examined by him at St. Bartholomew's Hospital. Mr. Pollock states that of 580 who were examined by him at St. George's Hospital, nine only contained fibroid growths. Braun and Chiari assert that out of 2,494 post-mortem examinations made in both sexes, in twenty-five instances were fibroid tumours found. The value of the two last statements in the estimation of the frequency of fibroid tumours is slight, inasmuch as the first refers to the condition found in women of all ages, and the second includes not only all ages but also the male sex. Though the statements of Bayle and of Klob are usually regarded as great exaggerations, yet there are no statistics which disprove them, while the results of Dr. West's investigations show that the growths under discussion are far from infrequent at all ages after puberty. It may fairly be concluded, not only that fibroid tumours are of common occurrence, but also that they exist in many instances without any troublesome or serious symptom. The course of fibroid t hen not interfered with and when left to nature is pretty wn; and this knowledge is of immense value, for it has sugge ds of treatment appropriate to certain grave conditions a course of the disease, which have in many instances prov Though not uncommon after the twentieth year, fibroid g or uterus have not been met with before puberty. They origi during menstrual life, while the uterus is active, between the twen and the fortieth or forty-fifth years, rarely if ever after the menopause. The period of their growth is also usually coincident with the period of uterine activity. This must be ascribed to the peculiar periodical changes which take place in the uterus during this period of life. There can be no doubt that a fibroid tumour lodged in the walls of a uterus partakes in some degree in the changes which take place in that organ, whether they be menstrual, senile, or the result of impregnation; and yet the morbid growth has a sort of independent existence. After the menopause they usually, though not always, cease to grow. During pregnancy they enlarge rapidly and become softer, and after delivery they may undergo involution just as the uterus diminishes in size. On the other hand, they may grow when the uterus is apparently quiescent. Their growth is often if not always in excess of the uterine enlargement. They sometimes continue to grow after the menopause; and after labour a fibroid growth may undergo involution to such a degree as to disappear altogether, or it may remain almost undiminished in size, or slough and be expelled in its entirety, or in fragments. What may be the relation of these changes in the uterus and in a fibroid tumour to the influence of the ovaries is somewhat uncertain, for considerable evidence has recently been brought forward to show that the periodical changes in the uterus are independent of ovarian influence. Though neither the dependence of menstruation issue, nor its independence of ovarian action has been established, yet there are reasons for believing that the presence of the ovaries has a considerable influence on fibroid tumours. Cases have been recorded in which the ovaries were removed with a view to relieve the hæmorrhage due to fibroid tumour and the operation was followed by markedly beneficial results, and such cases appear to shew that the presence of the ovaries favours one at least of the most troublesome and dangerous symptoms arising from fibrous growths.

Their rate of increase varies in different subjects and in the same subject at different times. This is a matter of observation and experience. The hard and little vascular growths increase slowly, while the softer variety grows more rapidly. In one case we have seen a fibroid growth, which was so small that it could only be suspected because of the profuse hæmorrhage present, and of a local induration of tissue felt immediately within the inner orifice after the cervical canal had been dilated by means of tents, attain the size of a walnut in a few weeks; while in another instance a tumour the size of an egg remained stationary for years. After the menopause they usually cease to grow, and often diminish in size; there are, however, numerous exceptions to this rule, in which growth proceeds rapidly after the change of life has taken place.

Fibroid tumours may undergo a variety of changes, some of which are of a temporary character, while others result in the arrest of their growth or in their complete removal. It is well known that the uterus gradually enlarges as the menstrual epoch approaches, and diminishes rapidly again during the flow. Fibrous tumours partake in this enlargement,—the increased flow of blood through the uterus causes an increased flow of blood through the tumour, and it increases in size and diminishes again when menstruation has set in. We have seen this menstrual increase in a fibroid growth which was lodged in the pelvis so marked as to cause complete retention of urine just before the appearance of the catamenial flow.

Fibroid tumours occasionally decrease in size permanently and without regard to menstruation. This appears to be due to the occurrence of œdema in the whole or a part of the growth and its subsequent absorption. Though the whole tumour may become œdematous, usually a part only becomes thus affected. When localised the fluid may or may not be surrounded by a cyst-wall. Paget says, "These formations of cysts are not rare in fibroid tumour, especially in such as are more than usually loose textured. It may be due to a local softening and liquefaction of a part of the tumour, with effusion of blood into the affected part, or to an accumulation of fluid in the interspaces of the intersecting bands; and these are the probable modes of formation of the roughly-bounded cavities that may be found in uterine tumours. But in other cases, especially in those in which the cysts are of smaller size, and have smooth and polished internal surfaces, it is more probable that their production depends upon a process of cyst-formation corresponding with that traced in the cystic disease of the breast and other organs."

Empty cavities are not infrequently found in fibroid tumours, and it is supposed that at one time such cavities were the receptacles of fluid. The fluid became absorbed, the cavities remained empty, and such an occurrence would readily account for the diminution in size occasionally observed in fibroid growths. In other instances the cysts are filled with blood or pus.

Occasionally these cysts form a considerable part of the growth, and the evidence of the presence of fluid in the tumour is apt to mislead and suggest the presence of ovarian disease, for tumours of the ovary are usually cystic, while those of the uterus are solid. A mixed cystic and fibroid growth of the uterus is called "fibrocystic." It is rare. It may attain a very great size, filling and distending the whole of the abdomen.

In some instances fibroid tumours undergo calcareous degeneration. They become hard, and have been described as bony. No true bone or cartilage is, however, ever found in them. The change consists in a deposition of the salts of lime and of other bases in their fibres, and is analagous to calcification of the arteries. This deposition may take place in their centre, or be scattered in an irregular manner through their substance. Sometimes it occurs in concentric laminæ, and when this happens in the superficial layers the growth becomes inclosed in a calcareous shell or covering. In other cases the whole tumour is involved in the degeneration. This is one of the modes in which nature cures or removes the tumour or arrests the progress of its growth. The calcified mass loses all vital connection with the uterus, and may be expelled by the vagina. This is the uterine calculus of old authors, the nature of which was first suspected by Dr. Matthew Baillie. The calcareous mass may, however, remain in situ, neither growing nor diminishing in size, nor giving rise to any symptoms.

It has been denied that fibroid tumours ever undergo atrophy and absorption; their hard and dense character naturally leads us to regard their disappearance in this manner as highly improbable; but the evidence bearing on this point shews that in some rare cases the disappearance of a fibroid tumour by this process really does take place. Several well-authenticated instances of such disappearance are on record, and its occurrence is now too well affirmed to be doubted. It appears to have been first observed in the unimpregnated organ by Sir Charles Clarke. The absorption of fibroid tumours during the process of involution following delivery has been repeatedly observed to take place by Scanzoni and Playfair.

Spontaneous detachment of a fibroid tumour may take place by the natural efforts of the uterus, by the expulsive action excited in the organ by the growth. Many, if not all, fibroid tumours originate in the substance of the wall of the uterus and are intra-mural or interstitial in position. Some of them remain in this situation throughout their course, while the position of others with regard to the surfaces of the uterus becomes altered. When they grow towards the mucous surface and project into the uterine cavity, they are called sub-mucous. When they grow towards the peritoneum and project into the serous cavity they are named sub-peritoneal. These three varieties of fibroid tumours, according to their position in the wall of the uterus—the sub-mucous, interstitial, and sub-peritoneal—it is of great importance to be able to distinguish clinically, because the treatment to be adopted, especially the surgical part of it, depends greatly upon the variety of the growth. There are two causes which play a part in bringing about a change in this position; one is the growth of the tumour itself. As it increases in size it necessarily projects in the direction of least resistance, that is, in that direction where it is covered by the least thickness of muscular tissue. If this be on the mucous side of it, the growth becomes sub-mucous; if on the peritoneal side of it, sub-peritoneal. This, however, is not the only agent, nor the most powerful, for the presence of the growth in the uterine wall excites contraction of the uterine fibres. A hard, solid mass, unyielding, denser than the tissue of the uterine wall, in which it is imbedded, forms an object favourable to the action of the womb. It gradually becomes pushed towards the mucous or serous surface, and there forms a protuberance. Frequently, indeed most usually, the expulsive process is carried but little farther, and the tumour remains partly imbedded in the muscular wall, partly projecting beyond its surface. Occasionally, however, the process does not stop at this stage, but the uterine action succeeds in extruding the offending mass out of the uterine tissue on to the mucous or serous surface, so that it becomes lodged in the cavity of the uterus, or in that of the peritoneum, being attached to the uterine walls by a pedicle only. The pedicle of the sub-peritoneal variety is usually more slender than that of the submucous. It consists almost entirely of connective tissue and vessels covered by peritoneum; while the pedicle of the sub-mucous variety is usually short, tough, and contains, in addition to connective tissue, a large quantity of muscular fibre cells. There is necessarily, from the manner of the production of pedunculated fibroid tumours, a stage in which they are sessile, and this condition may continue. When, however, the tumour has arrived in the cavity of the womb, muscular action becomes more violent and energetic, and the organ acts upon the foreign body at a greater advantage than when it was lodged in its tissues; expulsive actions are engendered and the mass is driven into the cervical canal, the walls of which gradually yield under the pressure from above. Ultimately the tumour may be driven through the cervical canal into the vagina. Meanwhile, the pedicle becomes elongated and more slender, and if it be sufficiently yielding, the growth may be expelled through the vaginal orifice, and even completely detached. This, however, is not a frequent termination. The pedicle is usually stout and unyielding, and offers great resistance, and the tumour remains in the uterus, or is driven down into the cervical canal or upper part of the vagina only.

Now and then, when the tumour is attached to the fundus of the uterus, and when the pedicle is unyielding, the contractions of the organ become so violent as to cause its inversion, partial or complete, and the tumour then lies in the vagina, the fundus uteri forming its base.

The sub-peritoneal variety is frequently pedunculated. Usually the pedicle remains short. Curious results may follow from the action of the abdominal organs upon a tumour projecting on the surface of the uterus into the peritoneal cavity. Professor Turner says, "Should a sub-peritoneal tumour be attacked by inflammation of its peritoneal investment and contract adhesions to its surrounding parts, it is placed in a position favourable to become separated from the uterus. This would be specially liable to occur if it became connected to a viscus, such as the bladder or rectum, which is continually undergoing changes both in size and position. The alternate dilatation and contraction of these viscera would necessarily exercise a considerable traction upon the tumour, which would tend to produce elongation of the pedicle; and ultimately, should the cause be sufficiently long in operation, complete detachment from the uterus. Even if the tumour were to attach itself to a fixed part, as the pubes or other portion of the pelvic wall, and the woman subsequently became pregnant, the growing uterus gradually rising into the abdomen might exercise such an amount of traction upon the pedicle as to attenuate it even to complete separation. The entanglement of the tumour between the walls of the small intestine which so frequently hangs down into the pelvic cavity, even though no distinct adhesions took place between them, would, during the peristaltic movements of the gut, exercise a certain degree of dragging upon it, especially if at the same time the pedicle became twisted. In those cases in which the tumours attain great size or great density, through calcareous degeneration, even without becoming adherent to adjacent parts, their own weight might probably assist in producing attenuation of the pedicle; but in estimating this as a cause productive of separation, we must always bear in mind the reciprocal pressure exercised upon each other by the walls and contents of the abdominal cavity."

The pedicles of sub-peritoneal tumours, though usually short, may acquire such length as to permit the growth to appear to float freely in the cavity of the abdomen, or to contract adhesions to distant organs as the liver or stomach. By separation of the pedicle the tumour may become actually free in the peritoneal cavity. In this condition it may remain perfectly innocuous, giving rise to no trouble of any kind, and the freely floating mass remains unchanged in structure. When a fibroid growth has contracted adhesions to other organs dangerous results may follow, for such adhesions may, by their contraction, or by peristaltic action of the intestine, constrict the gut and cause complete intestinal obstruction.

The sub-peritoneal variety is more prone to undergo calcareous degeneration than the other varieties of fibroid tumours.

Fibroid growths, even when lodged in the wall of the uterus, are liable to undergo a process of softening and disintegration. In this manner their whole substance may become a soft pulpy mass, or may be broken up into fragments and be discharged by the vagina or rectum. When expelled by the latter channel, adhesions form between the peritoneal surfaces of the growth and rectum, and the wall of the latter breaks down, making a way for the disintegrated tissue to escape into the bowel. Opinions differ with regard to the nature of the process. Sir James Simpson regarded it as sloughing—the result of inflammation. West on the other hand does not think that inflammation plays any part in bringing about the disintegration. "The process," he says, "seems to be one of death of the tumour, but the mode in which it is brought about is by no means clearly understood. It is not a process of inflammation nor of its ordinary results. The fibrous tumour when attacked by inflammation, presents a vivid rose-red colour, and shews a greatly increased vascularity, while local pain and the general signs of inflammation attend the process during the patient's life. The disintegration of the tumour on the contrary takes place unattended by symptoms which could lead to a suspicion of what is going on, and the outgrowth becomes soft and breaks down into a dirty putrilage." When a fibroid tumour has become pedunculated and lodged in the cavity of the uterus, the mucous membrane over its lower and most contracted part may become gangrenous. The whole tumour may ultimately become involved in the gangrenous process and be completely removed; or, instead of attacking the lower part of the growth, gangrene may involve the pedicle, and the mass become detached while still lying in the uterine canal, and then be expelled by uterine contraction.

It has been doubted whether fibroid tumours ever undergo cancerous degeneration. The general opinion is that it never happens, and there is every reason for believing that they have no tendency to become malignant, for there is only one case recorded where a fibroid growth of the uterus had become primarily cancerous. That case was described by Klob. He says that he placed in the Salzburg Museum a specimen of a fibroid tumour which had partially undergone cancerous degeneration, while there was no trace of cancer to be found in any other part of the body. If we accept this, we must admit the possibility of such degeneration, while we must also believe that such an occurrence is of infinite rarity. Fibroid tumours may, however, become involved in the progressive extension of cancer, for cancer may attack a uterus containing a fibroid tumour, and the latter may become involved as the former extends.

When a fibroid tumour has attained any size it always gives rise to some deformity of the uterus. The organ is enlarged and often displaced. The enlargement in some cases appears to be uniform, while in others it is not symmetrical. It is due to two causes, the presence of the tumour itself and a hypertrophy of the uterine walls. The amount of the latter varies much in different cases. It is greatest when the tumour is sub-mucous or interstitial, and least when it is sub-peritoneal. With the latter variety the uterus may become atrophied and be represented by a thin membrane only. The misplacement and deformity in shape of the organ may be of any kind, prolapsus or procidentia, elevation above the brim of the pelvis, version or flexion backwards, forwards, or laterally, or a twisting of the organ in such a manner that its canal is almost zigzag.

Fibroid tumours do not preclude pregnancy. Still, it is not common for impregnation to take place, and when it does it may lead to disastrous consequences. Abortion may take place, and very profuse hæmorrhage, or gestation may proceed to the full term, and natural delivery may prove impossible, and recourse to craniotomy or the Cæsarean operation become necessary. Should delivery be effected in the natural way, the patient has still great risks to run from post partum hæmorrhage, primary or secondary; from disintegration of the tumour in consequence of injury received during the birth, inflammation and pyæmia or septicæmia.

Sexual intercourse without impregnation appears in some cases to call attention to the presence of a fibroid growth in the uterus which had not been suspected before. The increased activity of the generative organs consequent upon marriage may give rise to hæmorrhage, and rapid growth of the tumour, and in some instances apparently to its destruction.

The causes of fibroid tumours are unknown. They are met with in every condition of life, in the married and the single, the fruitful and the barren. The negress is said to be specially obnoxious to these growths, while she suffers much less frequently from cancer than the white woman. Dr. McClintock believes that the scrofulous temperament is favourable to their production. They are frequently associated with neoplasms of the ovary. Sterility has been said to predispose to their formation, but there is no evidence to support this opinion, while there is no doubt that sterility is often entailed by their presence. Old standing menstrual disorders have also been credited with predisposing to their development, but when we bear in mind the slow rate of increase of these growths, we can hardly resist the conclusion that in persons who are the subjects of fibroid tumours, such disorders are the result of their presence while still small and unrecognised.

SYMPTOMS.—The symptoms of fibroid tumours vary greatly. There may be no symptoms whatever present, the patient being unconscious of anything wrong until she discovers an enlargement of the abdomen or a hard swelling above the pubes. This is more especially the case when the growth is of the sub-peritoneal form. When symptoms are present, and they usually are, there is no single symptom or combination of symptoms which is characteristic of the affection and which may not arise from other disease than fibroid tumour. There is generally a sense of weight in the pelvis and a dull aching in the sacral and lumbar regions. Menstruation is frequently, but not always, disordered. There may be severe dysmenorrhœa, and often there is hæmorrhage. The hæmorrhage may occur at the menstrual epochs only, and may be moderate in quantity, or be so excessive as to blanch the patient, to produce exhaustion, or even prove fatal. Frequently the menstrual bleeding is prolonged during a part of the inter-menstrual interval, and at a later period of the disease it may continue during the whole of the monthly period; or the catamenia may return every fortnight, or hæmorrhage of a profuse character may occur at irregular intervals without any regard to the epochs of menstruation. When the hæmorrhage is limited to the menstrual epoch it is called menorrhagia, when occurring at other than those times metrorrhagia.

The bleeding is evidently due to several causes, and much difficulty is found in explaining this part of the subject. It has been maintained that the source of the hæmorrhage was the surface of the tumour. In favour of this view it was urged that the bleeding ceases on the removal of the growth, or even on the application of a ligature to its pedicle; that the membrane covering the growth is highly vascular, and that the pedicle may contain vessels of considerable size. It has on the other hand been affirmed that the source of the hæmorrhage is the inner surface of the body of uterus, and in support of this view it has been stated that small growths cause as profuse hæmorrhage as large ones, that the most frequent form the hæmorrhage takes is menorrhagia, and that it is well known that the source of the menstrual blood is the interior of the body of the womb. Dr. West has brought forward one of the strongest reasons for believing that the hæmorrhage comes from the surface of the body of the uterus. He says, "A woman came under my care, who for three years had suffered from profuse hæmorrhages, which had ceased without known cause, for three months before I saw her. The non-appearance of the menses did not engage my attention as it ought to have done, and I accordingly excised a fibrous polypus the size of a small hen's egg, which grew by a short pedicle from the inside of the cervix. . . . For three years the polypus had irritated the womb, and blood had been abundantly poured out. Pregnancy took place, there was increased flow of blood towards the part, but no bleeding occurred." It is not exactly true that bleeding always ceases when the pedicle is tied, for several instances have occurred in which profuse hæmorrhage followed ligation. On the other hand the surface of the tumour is very vascular, and when injured may bleed profusely, and it does appear from this that the bleeding takes place from the whole of the mucous surface of the body of the uterus, whether it forms part of the covering of the tumour or no. In all cases the tumour is covered by mucosa, and there is no sufficient ground for supposing that any part of the mucous surface of the uterine body does not contribute to the hæmorrhage.

By reason of the growth of the tumour and the irritation caused by its presence, a greater quantity of blood than in health flows in the vessels of the uterus, and consequently the vessels opened during menstruation are larger than those opened at the same period in health, and permit a greater loss of blood. When the seat of fibroid tumour, the uterus is enlarged, its cavity is elongated, and as the hæmorrhage has its source in the whole of the surface of the body, it may be expected to increase in proportion to the increase in the area of that surface. There are many exceptions to this, and a very large fibroid tumour may be present while the menstrual flow is normal in amount. This is probably to be explained by, among other reasons, a lesser development of menstrual decidua. This I had an opportunity of verifying in a case of ovarian tumour, in which the cavity of the uterus measured four inches in length. Menstruation, though too frequent, was normal in amount. The patient died when menstruation was expected, and it was found that the decidual development for menstruation was extremely slight. From the congested condition of the inner surface, and the fact that the epoch was due, it was evident that menstruation was impending.

The position of the tumour in the uterine wall affects in a very marked degree the amount of the hæmorrhage. The sub-peritoneal variety frequently gives rise to no bleeding, while the sub-mucous generally gives rise to profuse bleeding. When again the tumour grows in the cervix, or at the junction of the body and cervix, the hæmorrhage may be very profuse, though the outgrowth be very small. This is probably owing to obstruction to the flow from the uterus caused by the presence of the tumour at that particular spot. The discharge is dammed back, the uterine cavity becomes distended, the potency of the bleeding vessels is maintained, and the hæmorrhage continues. This view of the case is favoured by the fact that when a free exit is made for the discharge by incision of the cervix, the menorrhagia often diminishes greatly or even disappears entirely for a time. Fibroid tumours may also cause hæmorrhage by exciting uterine contractions. The mucous lining of the uterus is soft, its vessels fragile. The tumour is hard and resistant. Contractions of the uterus upon such a body would inevitably lead to injury of the soft mucosa, rupture of its vessels and bleeding. It is not improbable that it is in cases of this kind anodynes act as hæmostatics. The causes of the hæmorrhage thus appear to be many and complex, but it appears to me that on the above principles the varieties in the amount of the loss in different cases may be explained. Hæmorrhage may be absent or moderate for a long time, and then become suddenly, and without apparent reason, profuse and dangerous. In seeking an explanation for such phenomena, the curious and singular effects of slight causes, as change of residence, sea-bathing, and others, upon menstruation when the uterus appears healthy, should be borne in mind.

Other discharges than blood are almost constantly associated with fibroid tumours. They are usually white or yellow, mucous or muco-purulent. They are sometimes watery or serous. They are usually odourless, but occasionally become as offensive as the discharge from a uterus affected with cancer, and may lead to a suspicion of the presence of that disease. Symptoms due to pressure on other organs often appear early. The bladder becomes irritable. There are frequent calls to micturate. The bowel is pressed upon, there are hæmorrhoids, frequent desire to go to stool and rectal tenesmus. Owing to the increase in weight, the uterus descends in the pelvis and may become prolapsed or procident. As the tumour grows, if it remain in the pelvis, the bladder and rectal symptoms may become urgent. There may be complete retention of urine owing to the pressure on the neck of the bladder. More rarely there is complete obstruction of the bowel, owing to its peculiar position in the pelvis. There are signs of pressure on the nerves of the sacral plexus, severe pain in the pelvis, numbness, and cramps in the legs; and of pressure on the veins giving rise to œdema of the lower extremities and vulva.

DIAGNOSIS.—From the rational symptoms it is not possible to recognise the presence of fibroid tumour of the uterus. These with a physical examination will, however, in the great majority of instances, suffice to establish the diagnosis. In some cases this is easy, in others very difficult, and in a few impossible. If the uterus be freely moveable, and the growth of moderate size, no difficulty will be met with. Immobility of the uterus on the other hand will occasion great difficulties. It may be confounded with pregnancy. Fibroid tumours which are likely to be confounded with pregnancy, usually give rise to hæmorrhage, and the tumour formed by them is harder and firmer than that formed by the pregnant uterus. When however the tumour is of the soft variety, the last-named character is not available. The changes in the vagina and the cervix will be of assistance, and it may be necessary to make an ocular examination with a view to detect any change of colour in these parts. Should all means fail, time will in such cases clear the obscurity. Pregnancy may be complicated with fibroid tumour. The want of uniformity in the enlargement of the uterus, or bulging at a particular part, and an undue enlargement for the period of pregnancy will give indications of such a condition.

When pregnancy has been excluded we may use the uterine sound with the view of measuring the length of the uterine canal, and the thickness of the uterine walls. Before using the sound, however, a careful bimanual examination should be made. This method of exploring the pelvic organ is most useful and trustworthy, and should in no case be neglected. By it we can in the majority of cases determine the size, shape, weight, contour, position, and movability of the uterus. In many cases such an examination alone is sufficient to establish the diagnosis. It is, however, not available in all cases, especially in those in which the abdominal walls are loaded with fat or the abdominal muscles are extremely rigid. When it fails the sound should be used.

From flexion of the uterus fibroid tumour may usually be readily distinguished by the combined method of examination alone, for flexion can readily be detected when the organ is held between the two hands. Should this method fail, the uterine sound should be introduced, the canal measured, and the uterus, if displaced, be restored to its normal position. If the tumour still remains when the uterus is replaced, it is probably a fibroid tumour. If it disappears, there may still be a fibroid tumour present, and the organ should therefore be examined by the bimanual method while maintained in position by the sound.

By a combined examination through the rectum and the vagina it may be ascertained whether the growth is situated in the anterior or the posterior wall of the uterus. The uterus should be drawn down as far as is consistent with safety, and the sound introduced into its cavity: the finger in the rectum can now estimate the thickness of the posterior uterine wall, and distinguish whether any enlargement behind the uterus grows from the uterus or from the pelvic wall. By a similar method the finger in the vagina can readily estimate the thickness of the anterior wall of the uterus, or the presence of any new growth in that situation.

Cases in which fibroid tumours situated in the posterior wall of the uterus have become fixed by adhesions to the peritoneum around present the greatest difficulty. It is sometimes impossible to determine whether the thickening be due to fibroid tumour, pelvic cellulitis, or pelvic peritonitis. When the history fails to make the condition in such cases clear, we must trust to time alone for the solution. It should be borne in mind that both fibroid tumour and pelvic peritonitis or cellulitis may be present together.

The diagnosis from ovarian tumour will be given when treating of that disease.

TREATMENT.—The treatment is both medical and surgical. In thi place we shall speak of the former only, the latter being beyond our limits. The medical treatment of these growths is chiefly palliative and consists in the prevention of hæmorrhage, the relief of pain, and the maintenance of the general health. In some rare cases, however, much may be done towards effecting a cure, and indeed cases are known in which fibroid tumours have completely disappeared during the administration of drugs. In the arrest of hæmorrhage, no therapeutic means is of greater value than rest. The rest should be absolute, physical and functional. The patient should remain in bed, and should not move out of it for any purpose. She should not get up to empty the bladder and bowels. Hæmostatics are often called for, as tannic or gallic acid, acetate of lead and opium, sulphuric acid, cannabis indica, vinca major, bitartrate of potash, alum, capsicum, oil of turpentine, sesquisalts of iron, Ruspini's styptic, digitalis, ipecacuanha, mercury. Any one or all of these when tried may fail, and then it may become necessary to plug the vagina and cervix uteri.

Acetate of lead and opium has answered well in my hands, but every now and then it fails; gallic acid or some other member of the group has, however, proved most effectual in other hands. When acetate of lead is administered, the patient should be carefully watched, for though usually no unpleasant results follow the administration of the drug, yet in some rare instances symptoms of acute lead-poisoning set in rapidly after a few doses of it have been taken. Should this occur, the administration of the salt should be at once stopped and Epsom salts given instead.

In some cases perchloride of mercury has succeeded when all else has failed.

With a view of effecting a cure several medicines have been in use. When treatment is undertaken with this view, it should be distinctly understood that the prospect of causing absorption of these growths is exceedingly slight, and that no treatment can be of any avail unless persisted in for a prolonged period. Iodide and bromide of potassium have been given with this object. The iodide is administered by the mouth and also applied in the form of an ointment to the abdomen and to the cervix uteri. Its effects are by no means of a satisfactory character. For some years the bromide of potassium has been much used. It is administered in ʒss. doses three times a day, and this should be continued for months. It is said to produce favourable results, but though I have tried it extensively, I have not been able to discover that it has had any effect whatever on the growth of the tumour.

Chloride of calcium is another remedy which has been given with a view to arrest the growth of fibroid tumours. It is given with a bitter or with a salt of iron if the latter be indicated, and its administration continued for months. There is a danger connected with its use. It is said to arrest the growth of the tumour by causing calcification of its arteries. Were this process of calcification limited to the tumour, chloride of calcium might be a very valuable remedy; the salt, however, acts in a similar manner on the vessels of the body generally, and thus brings about a condition more dangerous than the one for the cure of which it is given.

Ergot of rye is known to give rise to contractions of the muscles of the gravid uterus, and is supposed to have a similar influence over those of the unimpregnated organ. Ergotine—supposed to be the active principle of ergot—has during recent years been used in the form of injections under the skin for the cure of fibroid tumours, and some favourable results have been obtained from its use. Its disadvantages are that it gives rise to severe pain, redness, and inflammation, and often abscess at the seat of puncture. More recently a substance called sclerotic acid has been obtained from ergot, and it is said to be its active principle. It is soluble in water: six drops of water will dissolve one grain of the acid. The dose is half a grain, or three drops of the solution. It causes some irritation around the puncture, but less than ergotine. I have never seen abscess follow its use. I have used it in one case with marked results. A patient, aged thirty-five years, suffered from a large fibroid tumour, reaching three fingers' breadth above the umbilicus. She had continuous hæmorrhage, and was unable to move out of bed. The sclerotic acid was injected under the skin twice a week; the hæmorrhage ceased in the course of six weeks, and menstruation became regular in the course of a few months. The general health improved, and the tumour now reaches to the umbilicus only, and the patient is able to walk about, go up and down stairs with ease and comparative comfort. From a single case it is not possible to draw reliable inferences as to the value of the remedy, but it should be stated that when on two occasions the injection was suspended for a month, the hæmorrhage returned, and when recourse was had to the remedy again, the hæmorrhage ceased.

CYSTS.

Small cysts are not uncommon in the mucous membrane of the cervix; they are also met with in that of the body. They are the result of cystic degeneration of the follicles and utricular glands; Rokitansky, however, thinks that some of them are new formations. They are rarely larger than a hemp-seed, but occasionally attain the size of a small cherry. Their contents are fluid, viscid or caseous.

Cysts in the substance of the uterine wall are infinitely rare. Small collections of serum occasionally form under the peritoneum, but they are not surrounded by a cyst-wall.

Dermoid cysts containing hair and teeth have been found in the uterus.

True hydatid cysts have also been seen in the organ.

Carswell has figured a vascular erectile tumour of the uterus, and Klob has described a vascular outgrowth which he believed had its seat at the point of attachment of the placenta. The origin of the growth he refers to a paralysis of that part of the uterus to which the placenta was attached, and a consequent imperfect involution of the part; an explanation which, to say the least of it, is somewhat fanciful.

POLYPUS OF THE UTERUS.

All growths attached to the inner surface of the uterus or to the vaginal portion of the cervix, be they sessile or pedunculated, are called polypi. Malignant growths are, however, generally not called polypi, though they may assume a polypoid shape.

Polypi of the womb vary very much in structure, and are in consequence variously classified by different authors. Here they will be described under the following heads and in the following order:—1. Fibrous polypus; 2. Mucous polypus; 3. Fibrinous polypus; 4. Placental and vascular polypi.

Fibrous polypus of the uterus is much less common than fibrous tumour. Indeed it appears to be the ultimate condition of one of the forms of the latter—the sub-mucous. Fibrous polypus originates as a sub-mucous tumour. By its own growth and the contractions of the uterine wall, it gradually emerges from its bed in the substance of the organ in the manner described under Fibroid Tumours, and becomes lodged in the uterine cavity. It may remain closely attached to the wall of the womb, and then it is sessile or it may become stalked or pedunculated. It is of a round or pear-shape, the large end being directed towards the orifice of the uterus. Its surface is of a pink, deep red or purple colour, smooth, and sometimes presents small outgrowths, which give the mass an irregular shape. It is covered by a mucous membrane, which is a continuation of that lining the uterus, which bleeds freely when subjected to manipulation or injury. In structure the growth resembles fibroid tumour; it consists of fibrous tissue mixed with a varying proportion of muscular fibre. In some instances the growth is hard, in others somewhat soft and fleshy, and is then analogous to the softer variety of fibroid tumours. Occasionally cysts are found in its substance; it is then called fibro-cystic polypus. It varies in size, but never attains the enormous size that some fibroid tumours acquire. It may, however, be so large as to completely fill the pelvic cavity. In the great majority of cases it has its attachment to the body or fundus of the uterus. The latter is said to be its most common seat. Not rarely, however, it grows from the anterior or posterior wall of the organ. Occasionally, but very rarely, it originates in the cervix, and in some instances from the lips of the os uteri. The pedicle may be slender and fine, or thick, stout, and fleshy. It consists of connective tissue mixed with muscular fibre-cells from the parenchyma of the uterus, and blood-vessels. Sometimes it is formed by a duplication only of the mucous membrane passing from the uterus over the growth. Its blood supply varies much. The vessels are usually small, and the pedicle may be cut through with scissors without fear of hæmorrhage; but occasionally a vessel as large as a goose-quill supplies the growth.

The polypus, when lodged in the uterus, acts as a foreign body, excites contractions of the uterus, and gives rise to severe pain and profuse hæmorrhage. In consequence of the increased action of the organ, a hypertrophy of its substance takes place, and the uterus becomes enlarged, so that whenever a tumour of this kind has been removed, the wall is found thickened. The uterine contractions may, if the pedicle be slender and yielding, expel the polypus out of the cavity of the organ, or throw it off completely, as described when speaking of Fibroid Tumours.

If the pedicle be thick and flat, other and serious accidents may happen. When the growth is attached to the fundus, the tumour, driven down by the uterine contractions, may, on account of the unyielding nature of its pedicle, drag the fundus with it, and cause more or less inversion of the organ. This may proceed to such a degree as to cause complete inversion of the uterus and inversion of part of the vagina. The tumour as well as the fundus of the uterus would then be outside the vulva.

Fibroid polypus is liable to changes like those which take place in fibroid tumours. Inflammation, gangrene, fatty degeneration, and softening occasionally attack them, and these processes may result in their complete destruction, removal, and cure; on the other hand, absorption of the decomposed tissues may take place, and death follow.

As a consequence of inflammation, a fibroid may become attached to any part of the genital canal,—the vagina or uterus.

Calcareous degeneration, it has been said, takes place more rarely in the sub-mucous than in the sub-peritoneal variety of fibroid tumour. It is rarely seen in polypus.

Polypus does not absolutely prevent conception. Owing, however, to the irritation and inflammation set up by its presence, and to the mechanical impediment occasioned by it, especially when lodged in the vagina or cervix uteri, it is rare for conception to take place. When it occurs the pregnancy is associated with grave dangers. Abortion is not unlikely to take place during the earlier months; and should gestation run to the full term, as occasionally happens, danger is not over. Hæmorrhages frequently occur during its course, and after delivery profuse and fatal bleeding may happen. Should, however, the hæmorrhage be arrested immediately after the birth, if the tumour be left *in utero,* bleeding may occur again during the puerperal period, while if the growth be excised the patient runs a serious risk of pyæmia. For this reason, by universal consent, operations on the puerperal uterus are, if possible, avoided.

The chief symptom of polypus is hæmorrhage. In some rare instances this sign is altogether absent, however. It shows itself at first as excessive menstruation. There is menorrhagia. Later it occurs at any time, irrespective of the menstrual epochs. In quantity it varies greatly, and this variation appears to be independent of the size of the tumour. It appears to be more profuse when the growth is lodged in the cavity of the womb, and occasionally it ceases altogether when the polypus has been expelled into the vagina. The bleeding may be so great as to prove fatal.

There is leucorrhœa. The discharge may be purulent, mucous, or serous. It is usually odourless, or has but a slight, unpleasant odour. Occasionally, however, it is extremely offensive, and this is due to its retention in the genital passages and consequent decomposition. There are sacral and lumbar pains, and while the growth is lodged in the cavity of the womb severe expulsive pains, which cease entirely when the mass has been expelled into the vagina. The bladder is irritable: there is frequent desire to micturate; there may be incontinence; if the tumour be large it may press on the neck of the bladder and cause complete retention, or on the ureters, and give rise to hydronephrosis and uræmic poisoning. The rectum may be disturbed in its functions; there may be hæmorrhoids, mucous diarrhœa, or constipation from pressure. Like fibroids, they may press also on the nerves and vessels passing through the pelvis, and cause pains, cramps, and œdema in the lower limbs. Owing to the mechanical impediment offered by them to the exit of the menstrual and other discharges from the uterus, the Fallopian tubes may become dilated and regurgitation of the accumulated secretions may take place into the peritoneal cavity, or rupture of the uterus may occur in consequence of ulceration of its wall following pressure on it.

The general health of patients suffers variously, according to the amount of the hæmorrhage and leucorrhœa and their duration. Anæmia is often present. The complexion becomes sallow, earthy; the stomach gets out of order, the appetite is lost, the tongue is furred, and the bowels are constipated. Later on, nervous and hysterical symptoms are not infrequently present, and in some cases a feeling of great depression. When fibrous polypus is still lodged in the uterus, and the os remains undilated, the diagnosis is impossible. The uterus is enlarged and hard, and the presence of a sub-mucous fibroid may be recognised. If, however, the cervix be dilated by the efforts of nature, or artificially by tents, no difficulty will be met with in recognising the nature of the case. If the finger can be swept freely round the uterine cavity the size and attachment of the pedicle can be discovered. This can not usually be made out by means of the sound alone. If the lower part of the polypus ulcerates and becomes gangrenous, it may be mistaken for cancer, but a careful examination will reveal the lips of the uterus intact, and the pedunculated, fibroid nature of the growth. From mucous polypus it is distinguished by its hardness; it should be remembered, however, that true fibrous polypus has occasionally a soft texture. From sarcoma and cancer of the body of the uterus microscopic examination gives a certain means of distinction. Fibrous polypus, if left alone, may cause death by producing exhaustion as a result of frequent hæmorrhage and constant discharge, or as a direct result of profuse flooding, or by septicæmia in consequence of breaking down of the tumour. The prognosis is generally favourable, however, if the growth be removed. No treatment is of any avail until the tumour has been excised. When this has been accomplished the hæmorrhages and discharges cease, and the patient soon regains health.

Mucous Polypus.—Under this term are included those circumscribed growths which involve only the superficial layer of soft tissue on the inner surface of the uterus. These have received various names, depending to a certain extent upon their structure, and their relation to the uterine glands and follicles, as vascular, vesicular, cystic, channelled, tubular, and glandular polypi. They may grow from any part of the inner surface of the uterus. Some maintain that they rarely grow from the surface of the body, but this is evidently an error. Klob states that they are very frequently found in that situation, especially in the organs of aged women. I have met with such growths in the body of the uterus of a young woman, twenty-three years of age. In the cervix they may form simple projecting folds of membrane, or assume the form of sessile or pedunculated growths, from the size of a pea to that of a walnut. The follicles so abundant in the mucous membrane of the cervix uteri are not infrequently the subjects of enlargement and cystic degeneration, and take part in the formation of mucous polypi. The process may involve one follicle only, and this may attain the size of a pea, or a small bean; or a large number may be affected at the same time, and the canal of the cervix be filled by them. They remain partially in the wall of the uterus and project into the cavity simply by reason of their size, or they become dislodged, either alone or associated with an outgrowth of the mucous membrane, or become pedunculated and form a polypus. In the course of this process many of the follicles burst, and others then enlarge and take their place, but by rupture of successive follicles the mass may assume a harder and firmer texture, and its original structure be disguised. The process may affect any follicle in the course of the canal, or on the vaginal surface of the uterus, but more commonly those situated in the lower portion near the external uterine orifice are involved.

These growths are soft, smooth, round, or lobulated, and of a red colour. The pedicle may be slender, and of considerable length, even so long as to permit the tumour to hang outside the vulva, or short and stout; while in some cases they are sessile, and appear as simple outgrowths of the mucous membrane. They are covered by a layer of epithelium, columnar or tesselated, according as they grow from within or outside the uterus. On section the vesicular structure is readily seen. The vesicles may appear as small roundish cysts of various sizes, or as tubes or channels, having their course directed towards the surface. They usually contain a viscid, slightly yellowish fluid, and occasionally a little curdy matter. The pedicle is formed of connective tissue, bands of which spread between the vesicle, forming a sort of stroma, the proportion of which increases as successive vesicles rupture. In some cases the vesicular structure entirely disappears, and the stroma, with some cicatricial tissue, is alone left. Then the growth consists of a hardish mass, which may develope into a papilloma if the stroma continues to grow.

The growth may remain in the uterine canal, or it may be extruded into the vagina. In the latter event the pedicle becomes elongated, and the polypus may be ultimately thrown off. Very commonly mucous growths are sessile, and attached to the uterus by a broad base. Mucous growths of the body of the uterus are less common than those of the cervical canal, and they are far less frequently diagnosed. They occur in the single as well as in the married, in the young as well as the old, but more especially in women after the climacteric, who suffer from chronic inflammation of the uterus. They grow at any point of the cavity, but more often near the isthmus, or the orifices of the fallopian tubes. These growths are of various shapes, sometimes they form flattened elevations, or folds, on the surface of the uterus; in others they are round, or mushroom-shaped, and pedunculated. They usually project but a few lines only above the surrounding surface, but now and then they attain the dimensions of a strawberry. In large soft uteri which have undergone imperfect involution after labour or abortion, a peculiar granular condition of the mucous membrane is frequently found which may possibly be due to an imperfect removal of the old and effete decidua lining the uterus, the portions which remained having acquired a new life.

When projecting into the vagina mucous polypi are usually readily recognised. The speculum may be necessary for this, for they, by reason of their softness, may escape the finger. When in the cervix, and within reach of the eye, no difficulty can be met with in distinguishing them. When, however, they are in the body of the organ, and beyond the reach of the finger or sight, it is in many cases impossible to detect them. If a tent be introduced to dilate the cervix so as to admit of exploration of the uterine cavity, mucous growths situate in the upper part of the cervical canal, or just within the internal orifice, may disappear entirely, in consequence of the pressure exercised upon them, while if they remain unaffected by it they may elude detection by the finger by reason of their softness. When of considerable size a soft outgrowth can be readily recognised. If a portion of it be removed microscopic examination will distinguish between it and sarcoma of the mucous surface.

These formations are said to be the result of chronic catarrh of the mucous membrane.

The treatment is surgical, and consists in their removal.

The *fibrinous and placental polypus* are not outgrowths of the uterus, and therefore cannot be regarded as true polypi. The former appears to be either a coagulum of blood poured into and retained in the uterus, and become more or less decolourised and attached to the organ at a particular part; or an ovum which has attached itself to the uterus, and into which blood has subsequently escaped. The placental polypus consists of a portion of the placenta which has remained *in situ* after labour, and preserved its organic connection with the uterine wall. Their nature can be recognised only after removal, which should be effected as soon as the growths are discovered, for both varieties give rise as a rule to profuse hæmorrhage.

PAPILLARY TUMOURS OF THE UTERUS.

These may be benign or malignant. The malignant belong to the epithelial variety of cancer. The benign usually affect the vaginal portion of the cervix. They are generally small, but sometimes acquire a considerable size, and form the cauliflower excrescence of the uterus. The cauliflower excrescence, however, is more often malignant than benign in character. The growths may be few or many, simple, club-shaped, or branched. They are of a red colour, soft texture, and consist of the hypertrophied papillæ of the cervix; they are amply supplied with blood vessels, and are covered with a thick layer of epithelium. The symptoms are a serous watery discharge, sometimes stained with blood; irregular and profuse hæmorrhages may be present, in consequence of which the general health may suffer. They may atrophy and die, or they may undergo cancerous degeneration. This should be borne in mind when treating these growths. Their early removal when discovered, before any sign of malignancy makes its appearance, is imperative, and should be effected as far as possible from the seat of the growth, in healthy tissue.

Villous growths of the uterine canal are rare. I know but of three cases, two of which have been published, and the third was reported to me by Dr. W. R. Gowers. The first case was read before the Royal Medical and Chirurgical Society by Dr. Snow Beck in 1873 (*Proceedings*, vol. vii., p. 199). It occurred in a spinster aged fifty-nine years. Irregular hæmorrhages came on four years before death, together with a profuse whitish or yellowish discharge, without any previous symptoms of ill health. The discharge had at first no offensive odour, but towards the end it became very offensive. There was no ache or pain throughout. "The appetite became impaired, and ultimately extinguished, the tongue red coated and tremulous, the bowels always constipated, pain at epigastrium after eating, much flatulency, frequent sickness, distressing hot flushes, occasional giddiness, and sleepless nights. There was comparatively little loss of flesh, and nothing in the general appearance to indicate serious disease. She died with a sudden gush of hæmorrhage from the vagina.

"The uterus was somewhat enlarged, the cavities filled with a soft shaggy tissue, which hung down for about an inch and a half into the upper part of the vagina, and filled this portion of the canal. With the exception of a thin shell at the fundus, the whole of the contractile or muscular tissue of the uterus was converted into a soft shaggy tissue, which ceased abruptly at the external orifice, where the vagina begins. With the aid of the microscope the contractile fibre cells were seen slightly altered; lower down they contained in their substance a varying amount of pus globules; still lower down all form of the contractile fibre cell was lost, and, instead, collections of large-sized fat globules mixed with diffluent tissue were seen; and at the lowest portion it consisted of structureless diffluent tissue, mixed with numerous and various sized fat globules."

The case examined by Dr. Gowers was in some respects similar. It was that of a lady, over fifty years of age, who was the subject of progressive muscular atrophy. She was sterile and had had irregular hæmorrhages from the vagina and profuse serous discharge. During life no change could be discovered in the condition of the uterus. After death the organ was found to be somewhat enlarged, the os and vaginal portion of the cervix appeared to be healthy. The cavity of the uterus was filled by villi growing from the whole of its surface. They terminated abruptly at the internal orifice.

The third case was reported by Dr. Lusk to the Obstetrical Society of New York, and was published in the American Journal of Obstetrics, for January, 1878. The subject was a single woman, forty years of age, who had never been pregnant. Menstruation began in the fifteenth year and continued regular until the thirth-eighth. After that date it became profuse and painful. There was also a yellow watery discharge and expulsive pains. The canal of the uterus measured three and a half inches. The os uteri was small. It was incised, and a soft pulpy intra-uterine growth was detected and removed. It returned again. It was removed again, and again returned. Ascites appeared from cirrhosis of the cervix, and the patient died.

After death the uterus was found enlarged. The cervix was atrophied. The cavity of the body enlarged and filled with soft, shaggy masses, which sprang directly from the muscular walls. These were villi covered with epithelium of an almost cylindrical form. No trace of gland tissue or decidua cells were found.

Hæmorrhage, profuse discharge, and sterility were present in the three cases. Pain was absent in two, present in one. The vaginal portion was natural to the finger in the three, but in one the growth projected into the vagina before death. The youngest subject was forty years of age. The diagnosis could only be made by dilating the cervix with tents, and exploring the uterine cavity with the finger.

SARCOMA OF THE UTERUS.

Sarcoma affects the uterus under two forms. One—the recurrent fibroid tumour—grows from the parenchyma, like a fibroid polypus, and is attached to it by a broad pedicle. The other is a growth from the mucous surface, is sessile, and has a tendency to involve the whole surface.

The first variety, like fibroid tumour, grows usually from the body of the uterus and rarely from the cervix. It is softer than fibroid growths, breaks down readily when seized by the forceps, sloughs and disappears apparently altogether in consequence of injury such as the removal of a portion of it, but only to reappear again in the course of a few months. In structure it simulates fibroid tumour, that is, it consists of fibrous tissue and muscular fibre cells, but scattered through it are centres of round or spindle-shaped cells. The amount of the latter varies. They may form an inconsiderable part only, or, on the other hand, the chief mass of the growth. It may be expelled from the uterus like fibroid polypus. It grows rapidly, but usually does not attain a large size; occasionally, however, it forms a tumour reaching to the umbilicus. Secondary growths are sometimes found in the lymphatic glands and other organs.

The growths are rare, but may be met with at any age after twenty. Their causes are unknown. It has been supposed that in some instances they are degenerated fibroid tumours. Their symptoms are similar to those of sub-mucous fibroid. There are profuse hæmorrhages and serous or watery discharges. The discharge may be odourless or very offensive. During the early stages it is usually odourless. There are pains in the back and expulsive pains due to the efforts of the uterus to expel the growth; but there is no severe pain similar to that of cancer. Symptoms of pressure on neighbouring organs may be present if the tumour has attained a large size. The uterus is enlarged in proportion to the size of the growth. The os uteri may appear normal, but in many cases it is dilated and the tumour can be felt through it as a soft mass occupying the cavity of the uterus, or as a polypus hanging into the vagina.

The diagnosis is not easy. A soft, rapidly growing tumour with a broad pedicle, would lead us to suspect its true nature. It is incurable, but does not prove so rapidly fatal as cancer. Sixteen out of thirty cases died within twelve months from their supposed commencement, others have lasted for several years.

The treatment as far as medicine is concerned is that of symptoms only. We cannot affect the growth in any way except by removal and cauterisation of the pedicle, and though temporary relief may by this means be given, yet the tumour will sooner or later return and ultimately destroy the patient.

The second variety springs from the mucous surface of the uterus, has no distinct pedicle, and may affect a part or the whole of the uterine surface. It may attain a considerable size and grow out through the cervical canal, forming a large mass, filling the greater part of the vagina. It is of soft texture, its surface is uneven, and when of large size feels as if irregularly branched. In its later stages it undergoes ulceration, and the branched appearance may be due to removal of portions of it by sloughing. It can be torn by the finger without much difficulty, and in this manner portions may be removed for examination. The wall of the uterus may be destroyed by the pressure exercised upon it by the growth or be invaded by the morbid process. The organ, however, continues moveable, unless the growth becomes so large as to fill the pelvis, or pelvic peritonitis sets in and adhesions form. This form rarely affects other organs secondarily. It has been met with at all ages after fifteen. It always proves fatal, though early removal may retard its progress.

The symptoms are similar to those of the recurrent fibroid. The diagnosis can be made by microscopic examination only, discovering the structure of a small round-celled, rarely spindle-celled, sarcoma.

The treatment consists in attention to the general health and the alleviation of symptoms.

CANCEROUS TUMOUR OF THE UTERUS.

Fibroid tumour we have seen usually affects the body of the uterus, and rarely the cervix. Cancer on the other hand generally begins in the cervix and rarely in the body of the organ. It is found as a primary growth, but may occur secondarily to disease in the ovary. The scirrhous, colloid, medullary and epithelial varieties of the disease are met with in this organ. Scirrhus and colloid are rare; the medullary is the most common form and the epithelial occurs next in frequency. At one time scirrhus was believed to be a common affection of the uterus, and this was due to the error of confounding induration and chronic inflammatory conditions of the cervix with cancer. At that time scirrhus was believed to be a disease of slow growth lasting for years, which ultimately broke down suddenly and proved rapidly fatal. The distinction between the two conditions —chronic induration and cancer—is now universally recognised, though in some rare instances the diagnosis between them may remain doubtful for a short time. Paget has not seen a case of scirrhus of the womb. West has never seen an example in the dead subject, but met with it during life in one instance in 187 cases of cancer of the womb. Rokitansky states it is of extreme rarity, while Kiwisch affirms that it occurs three times in ten cases of cancer. The reason of this difference of opinion depends probably on the fact that the disease is rarely seen in its early stages. Klob says that "carcinoma of the uterus, in the majority of cases, occurs in what we might call a fibrous medullary form, that is, in the rare cases in which we are enabled to recognise and study the primary condition of the carcinomatous growth in the dead body we find that form which is described as fibrous carcinoma or scirrhus, whilst in those cases in which the disease proves fatal, we meet with the distinct medullary variety of carcinoma." There appears to be a tendency for the harder form of cancer to pass into the medullary as the disease progresses. Indeed this may be said of the other forms of cancer when they attack the uterus.

Medullary cancer of the vaginal portion grows in the substance of the part, the mucous covering not being at first involved. The lips of the uterus become thickened, hard, and tense. They may be uniformly swollen, or they may acquire a lobulated character. The thickening continues and the vaginal portion becomes greatly enlarged. The os uteri gapes, and the cervical canal becomes more capacious than in health. On section the affected portion is of a whitish grey or reddish grey appearance, firm, and the surface furnishes a little turbid fluid on scraping. The mass soon undergoes an alteration, it becomes softer and degenerates into a pulpy, soft, brain-like substance. The mucous membrane becomes involved in the diseased process, and is rapidly destroyed and "an ulcer forms, a ragged uneven sore, with raised irregular ragged edges; and a dirty putrilage, covering its uneven surface, takes the place of the smooth but enlarged lips of the organ, or if the disease go on still further the lips of the womb and its cervix are altogether destroyed, and a soft, dirty, white flocculent substance covers the uneven, granular and hardened substance which alone mark their former situation." (West).

The lips and cervix of the uterus having been destroyed, and the softened mass carried away, a large funnel-shaped cavity, having its apex at the internal orifice of the uterus, occupies the place of the os tissue. The mucous membrane of the cervical canal or portions of it may still remain after the removal of the softened tissue, and hang loose in the cavity like a polypus, or a cancerous outgrowth may project into the canal. While the process of softening is taking place the growth advances upwards, involving the cervical tissue: and outwards involving the tissues around, and every stage from the earliest form of cancerous infiltration to that of ulceration may be present at the same time. The disease appears often to be arrested at the internal orifice, but in time it extends beyond that point and involves the body and the fundus. The body of the uterus is generally enlarged as well as the cervix, and this has been ascribed to hypertrophy arising from an increased flow of blood through the part in consequence of the presence of the morbid process. Klob however maintains that the enlargement is in the majority of cases an early stage of carcinoma simply. Ulceration may commence on the surface and extend into the tissues of the cervix, more commonly however it is preceded by softening of the diseased mass.

Cancer may attack one lip of the uterus, or it may appear at first in the form of hard nodules, which grow and meet, and ultimately involve the whole of the cervix. The anterior and posterior lips are affected equally often. When ulceration has taken place the progress of the disease varies a good deal. In some instances it extends slowly, while in others it proves rapidly fatal. It spreads into the surrounding organs. Adhesions form between the lips of the uterus and the vagina, these together with the vaginal walls become involved in the morbid process and form a hard resisting mass. Commonly the upper third or half of the vaginal tube is involved, but in some cases the disease extends almost to the vulva. It may also extend upwards into the canal of the uterus and form warty or polypoid growths in the cavity of the body. Ulcers with or without a cancerous base also are seen in that situation. As the morbid process advances the tissues around the uterus are invaded—first the peri-uterine areolar tissue. This becomes thick and hard, and the womb is then surrounded by a ring of firm cancerous material. The peritoneum becomes inflamed and adhesions form between the uterus and the bladder and the rectum, and the whole of the pelvic organs become bound firmly together. The glands in the pelvis are usually, but not always affected, while those along the aorta are occasionally so. Later the disease invades the wall of the bladder, it spreads through the muscular coat, and ultimately attacks the mucous lining of the organ, and cancerous growths form on its surface. The mucous membrane is however not always affected, but is red, swollen, and inflamed. If the patient live long enough ulceration extends through the vesical wall and lays open the cavity of that organ, forming a communication between it and the vagina, and entails on the sufferer all the pain and discomfort arising from that condition, in addition to the, sometimes, terrible torture of cancer.

The rectum also may be involved in the disease. The growth extends along the posterior wall of the vagina and invades that of the bowel. Adhesions usually form between the surfaces of the pouch of Douglas, and as the ulcerative process advances and involves the wall of the bowel the cavity of the peritoneum is protected and remains usually closed. The mucous membrane of the rectum becomes affected and large masses of villous cancer sprout into the intestine. The bowel may however be laid open, and then if the disease has affected the bladder in a similar manner, the urine and the fæces are poured into the cancerous cavity occupying the place of the vagina.

Cancerous growths are sometimes found in the peritoneum. They form small tubercles—sessile or furnished with a slender pedicle. Accumulations of serous fluid like cysts are occasionally found between the bands of false membranes underlying the peritoneal surfaces.

The lymphatics, the oviducts, the ovaries, sheaths of nerves, cæcum or pelvic wall may become cancerous by extension.

Secondary deposits, which are less common than in cancer of other organs, have been found in the pelvic and lumbar lymphatic glands, the liver, lungs, ovaries, and most organs of the body.

The anterior wall of the vagina and the bladder are more frequently involved in the progress of the disease than the posterior wall and the rectum. According to West, the posterior lip is more often affected than the anterior.

The epithelial variety of cancerous tumour of the uterus attacks the cervix in the form of a papillary growth. The villi which are abundant on the vaginal portion become enlarged in a varying degree. The enlargement may affect the villi of a small part only, or of the greater part of the vaginal portion, as well as the upper part of the vagina itself. It is rarely that opportunity is had for an examination of this disease in its early stages. Dr. West has had two such opportunities, and I have had one. In my case the cervix was slightly enlarged, and somewhat harder than natural. Upon it were seen irregularly scattered small bright red fine soft villi, which bled readily when touched. In this case the villi did not attain a large size, but ulceration set in early and proved fatal in the course of a few months.

Dr. West says, describing the conditions he saw, "In the first case there was a patch of the size of a shilling on the surface of the posterior uterine lip, somewhat raised above the surrounding surface, of a vivid red colour, bleeding readily, and with a papillary structure so fine that it closely resembled the pile of red velvet. In the second case the disease occupied the anterior part of the anterior uterine lip, the outer edge of which felt sharp-cut and everted, and the speculum discovered on it a small patch of abrasion, soft and velvety to the touch, and seen under the speculum to be beset with fine sessile granulations."

In some cases the villi grow rapidly, become branched and form a large soft tumour, filling the upper part or the whole of the vagina. This is the cauliflower excrescence of the os uteri. The cauliflower growth may at first be benign, but is usually malignant. As stated when speaking of papillary growths, benign papilloma, whatever their size, may acquire a malignant character. Epithelial cancer of the cervix breaks down readily, and its base becomes ulcerated, the morbid process invades the neighbouring tissues, and afterwards runs a course similar to that of medullary cancer.

It is generally believed that the body of the uterus is not often the primary seat of cancer, though some authorities think this condition is not so very uncommon. There can be no doubt, however, that the disease is very rare, compared with the frequency with which cancer attacks the cervix. The form in which it is usually found in the body is the medullary, though the scirrhus and epithelial varieties are also occasionally seen there. It grows as one or more nodules or as a tumour in the walls, bulging into the cavity of the uterus, or on the peritoneal surface, or it may assume a papillary or polypoid form; or it may infiltrate the whole of the uterine walls. It breaks down readily into a soft pulpy mass, and the membrane over it becomes necrosed, permitting its escape into the vagina, peritoneum, bladder, or intestines. The growth extends beyond the uterus into surrounding organs, and adhesions form between the bladder, uterus, or intestines. The uterus is enlarged, sometimes greatly enlarged. The walls, when infiltrated with cancer, are greatly thickened, and the whole of the uterine cavity, to within a short distance of the os tissue, may be obliterated by adhesion of the opposite walls. When the growth is epithelial in character and originates in the surface of the mucosa the uterus is enlarged by expansion, but without marked thickening of the walls. The cavity may measure five or six inches in length. Cancer of the body of the womb is sometimes secondary to cancer of the ovary, or it may be the result of extension from the cervix.

CAUSES.—The cause of cancer is not known. It is more common in the female than in the male, in the ratio of about five to two, and this is due to the frequency with which the uterus and the breasts suffer. In about a third of all women suffering with cancer the womb is affected. The negress is said to be much less liable to cancer than the white woman; the reverse was found to be the case with fibroid tumour. Age has a marked influence on its frequency. It has been met with before puberty, but it is extremely rare before twenty-five. Its frequency then increases until the age of forty, and attains its maximum between forty and fifty, after which it diminishes; but still the disease has been observed at a very old age. It attacks the single and the married, but the latter much oftener than the former. Sexual excesses are said to favour its occurrence, but prostitutes are not particularly liable to the disease. Child-bearing seems to favour its development, for the average number of children to each fruitful marriage amongst patients affected with cancer is from 5·2 to 7, while the number per marriage in this country is estimated at 4·2. Hereditary taint has been traced by some in about 1 in 6; by others, 1 in 19; giving a mean of 1 in 12·6. Depressing emotions are said to predispose to the disease. It is a curious fact that certain orifices as the mouth, anus, pylorus, and os uteri are favourite seats of the affection.

Cancer of the uterus is rarely seen in its early stages, for at that time it may give rise to no symptoms of such a character as to alarm the patient.

SYMPTOMS.—There are no symptoms distinctive of cancer any more than of polypus or fibroid tumour, and this is especially true of the disease in its earlier stages. Indeed, in the womb just as in other organs occasionally, though unfortunately very rarely, it runs its course to an advanced stage without giving rise to any serious troubles. In other cases symptoms of a troublesome and even of a serious character may appear early, of which hæmorrhage, pain, and profuse leucorrhœa are the most frequent.

Hæmorrhage in a considerable number of cases appears to be the first indication of anything being wrong. It is this which attracts the patient's attention, and to this she refers the beginning of her illness. It comes on in a gush and may last a few hours or a few days and then cease for months or even altogether: or it may return at irregular intervals throughout the rest of the patient's short life. It may come on spontaneously or after exertion, and not infrequently during coitus. In other cases the hæmorrhage takes the form of profuse menstruation, this function being at first performed regularly as to time, but soon becoming irregular and frequent. In women in whom the catamenia have ceased, the hæmorrhage occasionally returns periodically and may lead to the belief that the monthly function of the uterus has been restored. In others, again, it forms a continuous drain, not profuse, and only sufficient to give the discharge a pinky colour. In the advanced and ulcerated stages of the disease, the bleeding is often very profuse and recurs at short intervals. Occasionally it is altogether absent at this period. The source of the hæmorrhage is the lining membrane of the body of the uterus, as is shown by the expulsive pains which not infrequently accompany it, its occurrence at an early period of the disease, and its occasional cessation when ulceration has taken place. There can, however, be no doubt that in many cases the ulcerated surface contributes to it in no small degree, inasmuch as profuse and alarming hæmorrhage occasionally follows after an examination instituted with every care and gentleness. Indeed, even in the early stages of the epithelial variety, before ulceration has taken place, considerable bleeding may take place from the highly vascular and delicate villi in consequence of examination made by the finger.

Pain in the sacrum and loins of a dull character is always present. The darting, lancinating pain, which is supposed to characterise cancer, is occasionally absent throughout the course of the disease. Pain is usually, though not always, absent during the first period of the disease, while ulceration and extension of the morbid process into the neighbouring organs is accompanied by severe suffering. Though more or less constant, it undergoes irregular and severe exacerbations, and the suffering becomes agonising. With the progress of the disease, attacks of local peritonitis occur accompanied by the pain peculiar to it. The pain increases in severity as the bladder is invaded, and urinary troubles are then superadded. There is occasionally pain in the ovarian region. As the disease invades the upper part of the vagina, and the nerves and vessels passing through the pelvis, severe pain and anasarca occur in the parts supplied as well, and sometimes that form of œdema peculiar to phlegnasia dolens.

There is at an early period a mucous, muco-purulent or watery discharge. The discharge is most profuse in the epithelial variety, and is then usually of a serous character, often stained of a pink colour. At first it has a peculiar and unpleasant, but not offensive odour. When ulceration has taken place it generally, but not always, becomes offensive, and sometimes so offensive that the patient is a burden to herself and to those around her. This is due in part to retention of the discharges in the genital passages, but chiefly to sloughing of portions of the cancer. Urinary troubles are almost constantly present; frequent, painful, or difficult micturition, and occasionally incontinence. If the walls of the bladder have ulcerated through, the urine flows into the vagina. Retention is rare. When the ureters are invaded they may become completely closed, the flow of urine into the bladder is prevented, the parts of the ureters above the constriction become dilated and tortuous, the pelvis of the kidney becomes distended, and the parenchyma of the organ destroyed by absorption or abscess. Occasionally there is extreme hydronephrosis. Nausea may follow, with convulsions, coma, and death. The convulsions and coma do not appear to be due in all cases to uræmia, for in some cases they disappear and the patients live for months afterwards. Extension of the disease to the rectum may give rise to obstruction of the bowel, or to catarrh, and serous diarrhœa. When the recto-vaginal septum is destroyed the fæces pass into the vagina.

Nausea and vomiting are frequent symptoms in the later stages. They depend on several causes, but are principally due to the disordered condition of the mucous membrane of the alimentary canal, resulting from the general cachexia. They are said to depend in some cases upon constriction of the ureters.

At the outset of the disease the general health may be good. Sometimes even where there is advanced ulceration the patient may be stout and healthy-looking. This, however, is very rare, for with the destruction of tissue, the hæmorrhage and other discharges, the pain and loss of sleep, a peculiar appearance, called "cancerous cachexia," is acquired. The blood becomes impoverished, the appetite is lost, the functions of the stomach and bowels fail, the pain prevents sleep, there is great thirst, the tongue becomes furred, then red, glazed, and aphthous. The body wastes, and the patient dies exhausted. This is the most frequent mode of death. Death may result however from hæmorrhage, convulsions, septicæmia, coagula in the heart in consequence of thrombosis in the veins of the cancer, peritonitis from perforation or extension of the growth into the peritoneum, or from some intercurrent affection.

In some cases of uterine cancer there is great sexual excitement, and the presence of the disease in the cervix does not unfortunately preclude the possibility of pregnancy. The occurrence of conception is one of the saddest and gravest complications that can happen to the subject of malignant disease of the womb, for the prospect of the birth of a living child is extremely slight, while the life of the mother will be inevitably shortened by profuse hæmorrhages, or by the process of delivery. The latter can rarely be accomplished by the efforts of nature, and artificial aid becomes necessary, and this usually in the form of perforation of the fœtal skull, or of the Cæsarean section.

The symptoms of cancer of the body of the uterus are similar to those of cancer of the cervix. There is profuse hæmorrhage, fœtid discharge, and pain. The pain comes on usually at an early period, while in cancer of the cervix it is often absent or slight until ulceration has set in.

DIAGNOSIS.—The diagnosis of cancer in its later stages is not difficult. The ragged, uneven ulcer with hard edges, bleeding with almost the slightest touch, the fixed uterus, the hardened tissue around the uterus pointing to the extension of the disease into neighbouring organs, shew only too clearly the nature of the case. In the early stages, when the disease is limited to the vaginal portion, when the uterus is freely moveable, there may be considerable difficulty, and this is the more to be deplored inasmuch as it is the time when accurate diagnosis is of the highest importance, for it is also the time, and the only time, for attempting, with a fair prospect, complete removal of the growth with the view of effecting a cure. The only condition for which it is likely to be mistaken is chronic induration of the cervix. In making the diagnosis the following points should be taken into account:—The history of the patient; the mean duration of cancer is about eighteen months, while chronic induration lasts for years, and is usually referred back to a difficult labour which happened long before. Hard nodules lying in healthy tissue point to cancer, the hardness of chronic induration is uniform. Chronic inflammation of the follicles of the cervix give rise to hard nodules in an enlarged vaginal portion; these can be distinguished by the speculum, and removed by puncture. The mucous membrane is fixed, and does not glide over the subjacent tissue in cancer. Spiegelberg has suggested the introduction of a sponge tent as a means of diagnosis. In cancer the canal will not dilate; the tissue of the cervix will not yield to the pressure of the tent as it does in chronic induration. Early in cancer the upper part of the cervix loses its defined character, and its limits cannot be made out owing to spread of the disease to the neighbouring tissues, while it can readily be defined in chronic induration.

COURSE.—A few cases of cancer of the uterus have re vered through the efforts of nature. The morbid mass has slough ay, and the disease has not returned. Some have recovered af the part has been removed by operation. These cases, however, a few in number, and the prognosis is usually only too certai disease is fatal. Its duration varies somewhat. The avera cording to West, Lebert, and Arnott, is about eighteen m according to Simpson, two to two years and a half; according to Barker, three years and eight months. The disease may run an acute febrile course, and prove fatal in four months; while, on the other hand, it may last ten or twelve years.

TREATMENT.—Medical treatment is only palliative. We cannot hope to arrest the progress of the disease. There is no drug which in any way affects its course. If discovered early, while still limited to the vaginal portion, complete removal may effect a cure; while in the ulcerated stage even, much may be done by surgical means to arrest its progress, or at least to render lighter for a time the intolerable burden of the sufferer, by arresting hæmorrhage and other discharges. Many surgeons, however, think that it is best not to interfere by operative means. The physician's power is limited to the relief of symptoms, and in this even he often signally fails. Functional rest should be enjoined. Marital intercourse should be interdicted because of the risk of hæmorrhage, and because the consequences, if pregnancy should follow, would be disastrous; it is moreover believed to favour rapidity of growth. Rest during the menstrual or bleeding periods is necessary. Should the hæmorrhage become profuse, astringents are useful. For the pain, anodynes should be administered in the smallest doses sufficient to give relief, beginning with hyoscyamus or belladonna; and as the suffering increases having recourse to opium and morphia. They may be administered by the vagina or rectum in the form of pessaries or suppositories, or by the mouth or under the skin.

For the discharges and fœtor strict cleanliness is absolutely necessary. Frequent ablutions and injections containing astringents and disinfectants as alum, lead, zinc, carbolic acid, Condy's fluid, or salts of iron are useful. The bladder and rectum symptoms should be treated as they arise. Great attention should be paid to the stomach and bowels. The diet should be regulated, and should not be too full, and stimulants should be given only in moderate quantities.

PELVIC HÆMATOCELE.

BY WILLIAM OVEREND PRIESTLY, M.D., F.R.C.P.

DEFINITION.—A tumour formed by the extravasation of blood into the pelvic tissues in the immediate neighbourhood of the uterus; and generally associated with some derangement of the menstrual function.

HISTORY.—Pelvic hæmatocele has only lately found a place in medical nomenclature, and until quite recently it was comparatively new to English medical practitioners. With the exception of a notice in Dr. Tilt's "Diseases of Women," and lectures published by Dr. West and Sir James Simpson respectively, British medical literature was a few years ago almost entirely barren on the subject, and few cases had been recorded. The first clear and intelligible account of the affection was published in France, by M. Nélaton, and after the original observations by M. Nélaton on the subject, it furnished a topic in France for numerous theses, reports, and discussions. Hæmatocele is probably not more frequent in France than in England, for since attention was attracted to the subject, experience has proved that it is of common occurrence in this country as elsewhere; Dr. Barnes has especially drawn attention to its frequency. A numerous array of instances have been chronicled, and many British practitioners have written concerning it, or have made it the subject of discussion in debating societies.

Some of the earliest instances, where the recorded particulars leave no doubt as to the identity of the disease, occurred in the practice of M. Récamier in the Hôtel-Dieu in Paris. One of these was published in the *Lancette Française* July 21, 1831, under the title "Tumeur sanguine du Bassin;" a woman 28 years of age after a miscarriage, had a large tumour in the true pelvis behind the uterus which projected into the vagina, M. Récamier believing it to be an abscess, opened it, but instead of pus, only dark half-coagulated blood escaped from the aperture. In 1841 M. Bourdon in the *Revue Médicale* described the physical signs of blood tumours situated in the peri-uterine cellular tissue of the pelvis, and somewhat later M. Velpeau, in his "Mémoire sur les Cavités Closes" published additional cases, and was evidently acquainted with the true character of these affections. Others were reported by MM. Bernutz and Piogey. M. Bernutz claims priority in having pointed out in 1848 the relation between pelvic blood tumours and disturbance of the menstrual function, but he states that in his opinion the honour of having first discovered true hæmatoceles belongs to Ruysch in 1691. To M. Nélaton, the distinguished Professor of Clinical Surgery in Paris, belongs the merit of bringing this affection into such prominence that henceforth it takes a permanent place in our nosology. In 1850 M. Nélaton drew the attention of his class to the occasional occurrence of fluctuating tumours situated between the uterus and rectum, which, on being laid open, were found to contain extravasated blood. From the position of the tumour he gave it the name of "retro-uterine hæmatocele," a title still applied to it by some authorities, but apt to be misleading, inasmuch as hæmatocele is found in other localities in the pelvis, besides the posterior aspect of the uterus. In 1851 M. Nélaton made retro-uterine hæmatocele the subject of clinical lectures in the hospital, and these were afterwards published in the *Gazette des Hôpitaux*. The description there given is clear and precise, and without detracting from the merits of those who preceded him, it may be said that until M. Nélaton made it a subject of clinical instruction, it was absolutely unknown to the majority of medical practitioners, both in France and elsewhere. It was only in 1850 that M. Malgaigne is reported to have attempted the enucleation of a supposed fibroid tumour of the uterus, which proved to be a collection of blood, and the operation was followed by fatal hæmorrhage. The lectures of M. Nélaton having fairly roused attention, and shown that the disease in question was by no means so rare as might have been supposed from the little which had been said and written on the subject, several *brochures* and books speedily followed. Among the first and best of the theses on hæmatocele was that of M. Vigués a pupil of M. Nélaton, and later followed those of MM. Fénerly, Voisin and others. In 1860 M. Voisin published an octavo volume on "Retro-uterine Hæmatocele and Non-encysted Extravasations of Blood in the Peritoneal Cavity of the Pelvis," and further contributions were made in France by Laugier, Rouget, Fénerly, Puech, and MM. Bernutz and Goupil. In Germany by Virchow, Scanzoni, Braun, Herber, Credé, Breslau, Seyfert, and Olshausen; and in this country by Drs. McClintock, Matthews Duncan, Barnes, Tuckwell, Meadows, and Madge.

NOMENCLATURE AND PATHOLOGY.—The affection has received various appellations, "retro-uterine hæmatocele," "peri-meterine or peri-uterine hæmatocele," "hæmatoma," "pelvic thrombus" and "pelvic hæmatocele." The last seems to be the most comprehensive, and may be employed so as to include all the forms of tumour in the true pelvis formed by extravasated blood, irrespective of locality and apart from the causes or from the several theories which have been promulgated as to the way in which they are produced, always premising that they have their origin in the organs of reproduction. Much discussion indeed has taken place concerning the true definition of hæmatocele. M. Nélaton and his followers grouped together under the term "retro-uterine hæmatocele" all the varieties of blood tumour found posterior to or around the uterus, irrespective of their causes. M. Voisin restricts the term to those cases, in which blood is extravasated *into* the peritoneal sac between the uterus and rectum, and further, according to him, the result must be due to some accident of menstruation. M. Bernutz, one of the earliest and most authoritative writers on the subject, insists that M. Nélaton's grouping is irrational, and that uterine hæmatocele is not a specific disease, apart from that which caused it, but is simply a hæmorrhage symptomatic of certain morbid conditions, which ought to be the main object of pathological study. Endeavouring to define true hæmatocele, M. Bernutz points out the analogy between the *tunica vaginalis* in the male, and the utero-rectal *cul de sac* in the female, the difference between the two only being that the folds of peritoneum forming the *tunica vaginalis* are external to, and shut off from the abdominal cavity in surrounding the testicle, while in the female the analogous folds of peritoneum subtending the two ovaries together form an open sac communicating with the general cavity of the peritoneum above. Just therefore as he would apply the term "hæmatocele" in the male to a collection of blood in the tunica vaginalis, he restricts the appellation in the female to collections of blood in the retro-uterine pouch of peritoneum, and referring to the *causes*, to those blood tumours only which arise from some accident of menstruation. In Great Britain the term "hæmatocele" in the female is used in a wider and more comprehensive sense, and includes tumours formed by the extravasation of blood not only into the retro-uterine *cul de sac* of peritoneum, but elsewhere around the uterus and especially into the cellular tissue in the pelvis, which lies outside the peritoneum. Derangement of the menstrual function is recognised as one of the most fertile sources of pelvic hæmorrhage, but other causes also are admitted. Dr. McClintock in an able paper on this subject remarks that he "cannot at all agree with M. Bernutz, that to discover the existence of pelvic hæmatocele constitutes only the half, and the less important half of the diagnosis: on the contrary, it is, I should say, by far the most important half; for if we overlooked the hæmatocele, and were cognizant only of the morbid condition from which it had arisen, what errors of prognosis and treatment might we not commit?" As a practical fact it may indeed be pointed out that the treatment of effusions of blood into the pelvis must in a much larger degree be influenced by the rapidity, extent, and position of the extravasation, than by the pathological condition which caused it, and that although M. Bernutz is doubtless correct in his assertion that the sanguineous effusion is only a symptom and effect of some pre-existing pathological condition in the same sense as menorrhagia may be, yet all M. Bernutz contends for would for all practical purposes be attained, by steadfastly bearing in mind that like menorrhagia or uterine hæmorrhage it may proceed from a diversity of pathological causes.

Again, much controversy has arisen concerning the anatomical situation of the tumour in cases of hæmatocele. M. Voisin and M. Bernutz only admit those cases to be true hæmatocele in which the blood is poured *into* the peritoneal sac between the uterus and rectum. But according to these authors' own showing, instances do occur where blood is extravasated into the cellular tissue around the uterus, and beneath and outside the cavity of the peritoneum. These, however, are rather regarded by them as cases of "thrombus," being akin to those blood tumours which are found occasionally in the external genitals, in connexion with the puerperal state, and may be produced by violence, and influences other than those associated with menstruation. It may nevertheless be reasonably argued that it is an unwarrantable violence to separate so far apart these two forms of pelvic blood-swelling as to give them distinct appellations. Both have their position deeply situated in the pelvis; both arise from the rupture of, or escape of blood from vessels supplying the organs in the pelvis; and in both, if the extravasation be sufficiently sparing and slow, the blood becomes encysted. The family resemblance is further borne out by the tendency in both cases to the appearance of the tumour about the time of a catamenial period. It is quite true that the ovaries, the Fallopian tubes, and the uterus are the organs principally engaged in the menstrual act, and any escape of blood from these internally is most likely to flow *into* the cavity of the peritoneum; but it must also be remembered that during menstruation, and especially at its commencement, the whole uterine system becomes more vascular, the circulation in the broad ligaments is increased, the hæmorrhoidal vessels become distended; indeed, all the pelvic organs receive an increased supply of blood, and the abdomen itself becomes fuller. M. Rouget has described an intricate and tortuous plexus of vessels lying just beneath the ovary in the folds of the broad ligament, which, during menstruation and other analogous conditions, become so distended as to form a sort of erectile organ. This he has termed the bulb of the ovary. A condition favourable to the escape of blood exists therefore in all the pelvic tissues when the catamenia recur, and although the mode in which hæmorrhage takes place into the retro-uterine *cul de sac* of peritoneum may be more obvious than into other situations, yet there is ample evidence to show that blood is occasionally extravasated into the pelvic cellular tissue in such quantities as to form a considerable tumour. Post-mortem examinations have not been so frequent in these cases as to afford any very extensive data to illustrate this point, for there is no doubt that in the fatal cases the extravasation is more frequently intra-peritoneal. Dr. West, in his first edition of "Diseases of Women," has collected the records of eight autopsies, and of these we find that in two the blood was poured out behind the uterus and *beneath* the peritoneum; in one beneath the peritoneum in the iliac fossa; and in another between the folds of the broad ligament.[1] Sir James Simpson has published an account of a post-mortem examination, where the blood tumour was unmistakeably beneath the peritoneum, behind the uterus, and a diagram shows the manner in which the serous membrane was raised up to form the roof of the cyst. In another of Sir J. Simpson's cases, one of the upper hæmorrhoidal vessels had given way, and produced a blood tumour in front of the rectum. Dr. Matthews Duncan has convinced himself that the extra-peritoneal form is probably a common form of the disease, although he admits that the effusion is intra-peritoneal in many cases. Dr. Tuckwell has collected 41 cases where post-mortem examination was made. Of these the extravasation of blood was intra-peritoneal in 38 cases; an account which, however, only proves that the intra-peritoneal form is the more fatal, as previously observed. The opinion that hæmatocele may be extra-peritoneal as well as intra-peritoneal, is shared by MM. Huguier, Nonat, Robert, Becqueril, Verneuil and Prost; M. Nonat, after a careful study of this affection in La Pitié and elsewhere, states in his systematic work on the diseases of the uterus, that he believes the extra-peritoneal form is more frequent though less grave than the other, and he believes it possible to diagnose the two varieties and prescribe appropriate treatment for each. Be this as it may, it seems desirable to make the definition of hæmatocele sufficiently wide to embrace all the forms of tumour resulting from the extravasation of blood around the uterus, whether it be inside or outside the peritoneum, and without reference to the causes producing it, so long as these causes are connected with the generative organs. Certain varieties may be more frequently associated with the pregnant and puerperal states than with menstruation, but practically they are attended by the same train of symptoms and admit of little variation in treatment.

In studying the anatomical positions of hæmatocele it is necessary in the first instance to note concerning the intra-peritoneal variety that there are two forms which differ not in the causes or sources of hæmorrhage, but in the abundance or rapidity of the bleeding from whatever source it comes. Thus if hæmorrhage be abundant and rapid, no localised tumour is formed, but the blood spreads itself over a large surface of the peritoneum, and if the patient does not sink from loss of blood, death results speedily from the extensive inflammation produced. This has been called "non-encysted hæmatocele or extravasation." On the other hand, if blood be poured out in limited quantity and sufficiently slowly, it commonly drops down into the recto-uterine *cul de sac*, and there becomes encysted, being ultimately surrounded on all sides by lymphy adhesions. The way in which blood becomes encysted to form hæmatocele in the retro-uterine space is thus described by M. Voisin:—

"When blood escapes from the ovaries, the tubes, or the uterus, it falls naturally behind the broad ligaments into the retro-uterine peritoneal space, limited before by the broad ligaments and uterus, behind by the rectum and lateral folds of the peritoneum,—on all sides by serous membrane. Above, the *cul de sac* is open, and communicates largely with the rest of the abdominal cavity. In some rare cases the blood is carried in part into the vesico-uterine space, but in a very small proportion compared with the mass extravasated behind the uterus. Hardly have some drops of blood penetrated into the serous cavity than it inflames. This inflammation results in speedily establishing adhesions between all the pelvic organs, or rather between their peritoneal coverings. The coils of intestine are pushed upwards by the extravasated fluid, or rise upwards by their own lightness. The collection of blood encysts rapidly, thanks to the energy of the inflammation of the serous membranes and the formation of cellular adhesions. The sides of the tumour are then limited, before by the broad ligaments, behind by the rectum and peritoneum, below by the recto-uterine *cul de sac;* above, by the coils of intestines which, by their adhesions to the fundus uteri, the broad ligaments, the ovaries, the tubes, the round ligaments, and the peritoneum which covers the lateral parts of the pelvis, form for the cyst a sort of resisting roof."

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[1] These statistics are not inserted in the third edition, and Dr. West there submits to the authority of M. Bernutz on this point.

In the "extra-peritoneal variety," the blood is poured out into the meshes of the cellular tissue which surrounds the uterus and other organs in the pelvis. In these cases the tumour is much less constantly situated between the uterus and rectum, than in the variety last described, and it may, indeed, be formed in any part of the pelvis where rupture of a vessel has taken place, and where the cellular tissue is lax enough to permit separation of its layers. It divides its frequency between the posterior aspect of the uterus, and laterally between the folds of the broad ligaments. Here the vessels are most numerous, have the largest calibre, and being surrounded by looser tissue than elsewhere, are more liable to unequal degrees of distension. If the extravasated blood be considerable in quantity and the tumour large, the peritoneum will be separated from the structures upon which it normally lies, and either pushed aside, or raised upwards towards the cavity of the abdomen. In both the intra- and extra-peritoneal forms, the position, shape, and dimensions of the swelling vary in accordance with the exact situation of the vascular rupture and the amount of blood effused.

From the results of clinical observation it is highly probable that small extravasations of blood frequently take place in certain subjects about the time of the catamenial periods, but inasmuch as the quantity effused is too sparing, to form a well defined swelling, so the symptoms, though the same in kind, are commonly more obscure. When a distinct tumour is formed, it will occupy a space in the pelvis in proportion to its bulk, and may, if large enough, be felt through the abdominal walls cropping up out of the pelvis, or if smaller only by vaginal examination. The pelvic viscera are necessarily pressed upon and displaced in most cases where the swelling occupies the cavity of the pelvis proper and attains any considerable size. Commonly the uterus is pushed so far upwards and forwards that the cervix can with difficulty be reached above the pubes, while the tumour is found occupying a large space in the posterior half of the pelvis.

The *sources* of the hæmorrhage which have been described, are several. Voisin considers only three, viz., congestion and hæmorrhage from the vesicles of De Graaf during a menstrual period; reflux of blood from the uterus into the Fallopian tubes and peritoneum; and hæmorrhage originating in the Fallopian tubes themselves. Bernutz discusses five sources or causes, and classes the varieties in accordance with the causes thus:—

1. Hæmatocele symptomatic of rupture of utero-tubar varices. 2. Hæmatocele symptomatic of blood exhalation from the pelvic peritoneum. 3. Hæmatocele symptomatic of rupture of the ovary or Fallopian tube. 4. Hæmatocele symptomatic of difficult menstrual excretion. 5. Hæmatocele symptomatic of excessive secretion from the genital organs—metrorrhagic hæmatocele.

The evidence concerning some of these sources of hæmorrhage is indistinct and inconclusive, while the frequency of others is sustained by accumulated observation and testimony. The causes may be conveniently studied under the following heads.

1. There would seem to be no doubt that rupture of a vessel in some of the structures of the ovary is a frequent cause of pelvic hæmatocele. Physiological investigation has fully proved that at each ovular period which corresponds in the human female with the appearance of the menstrual discharge, one or more Graafian vesicles near the surface of the ovary become distended with crimson contents and at length rupture, and discharge themselves into the infundibular extremity of the Fallopian tube. A certainty quantity of blood escapes at the time, but under normal conditions the amount is small. When, however, any antecedent morbid change has altered the structure of the ovary and increased the size of its blood vessels, the blood effused may be much more considerable in quantity. Congestion and hypertrophy by enlarging the calibre of the vessels, induce a tendency to unusual hæmorrhage at the periods of ovulation, and the same may be said of cystic disease of the ovary. It is by no means uncommon in the post-mortem room to find small collections of extravasated blood in the substance of the ovary, and occasionally ovarian cysts are met with filled with coagulated blood, which has been poured into their interior, from the rupture of some of the large vessels ramifying in the parietes of the cyst. Death has been known to result from intra-cystic hæmorrhage of the form just mentioned. It can be readily understood how over-distention of a cyst or congeries of cysts in the ovary, more particularly if the effects of any accident or violence be superadded, might lead to rupture of the cyst-wall and extravasation of blood into the peritoneal sac. M. Voisin adduces several examples of hæmatocele produced in this way, and arrives at the conclusion that there is usually some pre-existing disease of the ovary which pre-

disposes to laceration of the blood vessels and consequent extravasation. M. Gallard proposes a modification of this theory of ovarian hæmorrhage, and suggests that in some cases hæmatocele is due to the presence of an ovum which has missed the oviduct and dropped into the peritoneum.

2. The Fallopian tube—the mucous membrane of which contributes to the menstrual flux—would seem occasionally, when unusual excitement or congestion exists, to be capable of pouring out so large a quantity of blood from its free extremity, as to give rise to hæmatocele. In like manner, blood may, if retained in the uterine cavity by occlusion of the os, or from displacement of the womb, as in extreme retroflexion, be driven along the oviducts to the peritoneal cavity, or burst the tube and consequently form hæmatocele. Dr. Matthews Duncan has pointed out that obstruction or occlusion of the os uteri is not necessary to make blood secretion in the cavity travel along the Fallopian tubes and so enter the peritoneum. He states that besides the tubes being periodically dilated to permit the passage of ova, they are sometimes in a continued state of dilatation and patency from pathological conditions. Under these circumstances even when the os uteri is sufficiently pervious, the mechanical arrangements of the viscera and the ærostatic mechanism of the abdomen walls, will drive fluid along the tubes and so explain the production of hæmatocele.

3. Puech, Voisin, and Bernutz have drawn attention to the rupture of a vessel in the utero-ovarian plexus lying between the folds of the broad ligaments as one occasional cause of hæmatocele. In certain patients the veins here, as well as in the lower extremities, and round the vulva and anus, are apt to become varicose. In this condition, their coats are thinned and weakened and prone to give way under increased pressure from muscular efforts or other causes. Probably an extra-peritoneal hæmatocele would be produced by such rupture, but M. Voisin states that in all cases of this kind which have been recorded, where laceration took place into the peritoneal cavity, the loss of blood has been so rapid and profuse, that no time has been allowed for it to become encysted, and immediate death has been the result.

4. M. Tardieu, M. Bernutz and others have described instances in which the source of bleeding was the general surface of the peritoneum affected by disease somewhat analogous to hæmorrhagic pleurisy. This has received the name of "hæmorrhagic pelvi-peritonitis." Virchow and Herber suggest the possibility of capillaries being formed in false membranes over the pelvic peritoneum, and being the source of bleeding in these cases. Professor Dolbeau who gives his adhesion to this theory asserts that an immense number of cases of retro-uterine hæmatocele are produced by pelvic peritonitis of a hæmorrhagic form, and that this explains the less serious nature of some cases as compared with those having a tubal, ovarian, or varicose source.

5. A further source of internal hæmorrhage giving rise to hæma-

tocele, which has been described, is a general hypersecretion of blood from all the genital surfaces, internal and external, to which M. Bernutz has given the name of "metrorrhagic hæmatocele." This pathological condition, which is not like the last form, the result of inflammatory action, is somewhat analogous to the hæmorrhagic diathesis. The formation of hæmatocele internally is preceded and accompanied by excessive menstrual discharge from the uterus and vagina, and it is presumed that a simultaneous bursting forth of blood takes place, both from the mucous surfaces of the genital canals, and the peritoneum covering the outer surface of the uterus and its appendages. This class of cases has especially been noticed during the progress of the eruptive fevers, and a diminution in the amount of fibrine in the blood has been supposed to favour the hæmorrhage. Scanzoni has related the case of a patient affected with measles, who died immediately after the commencement of menstruation, from hæmorrhage which took place into the peritoneal cavity from the left Fallopian tube; and M. Bernutz has collected many examples under this head which he has classed in groups according to certain characteristics or differences. Belonging to this order are hæmattoceles characterised by some cachexia, or by anæmia and chlorosis, in which the blood is impoverished and thus more easily escapes from its containing vessels.

Although it is well established that hæmatocele takes place most frequently at or about the time of the catamenial period, and for the reasons previously stated is then the result of some derangement of the menstrual function, yet the affection occurs in some instances where the catamenia are absent and the function of spontaneous ovulation is suspended. During pregnancy and after delivery and abortion, extravasation of blood both into and outside the peritoneum may give rise to the formation of a pelvic blood swelling, having all the characters commonly observed in a typical hæmatocele. Examples of this kind have been recorded by Dr. West, M. Voisin, and M. Bernutz.

MORBID APPEARANCES.—The reports of post-mortem examinations in cases of non-encysted blood extravasation into the peritoneum, are proportionally more numerous than those of encysted hæmatocele, inasmuch as the former much more frequently ends fatally. (On these portions of the subject we quote largely from M. Voisin.) In the non-encysted form it is generally found after death that the skin of the body is devoid of colour, and the belly tumid, more particularly in the region of the hypogastrium. Black fluid blood may escape in considerable quantity when the abdomen is laid open. The intestines are distended with gas, and pushed up above the mass of blood contained in the pelvic cavity. The abdominal organs are often covered with clots, the intestines stained of a bluish colour, and in one recorded instance the mesentery was infiltrated with blood. The amount of blood, fluid and coagulated, contained in the pelvis and abdomen has repeatedly been found to be as much as four pounds. In twenty cases quoted by M. Voisin, the source of hæmorrhage was traced in sixteen to some distinct lesion. In six cases the hæmorrhage came from the ovary; in four from the rupture of an ovarian varix, in two from the cavity of the uterus, and in four from the Fallopian tube. In the remaining four no distinct lesion could be found, and it was supposed that the hæmorrhage resulted from an exhalation or oozing of blood from the surface of the peritoneum.

In the few autopsies which have been made of women who have died with limited or encysted hæmatocele, no great tumefaction of the abdomen was met with. Commonly the general surface of the peritoneum was healthy, except that occasionally adhesions were remarked between the intestines. If any of the adhesions forming the boundaries of the cyst had been torn so as to allow the cyst contents to escape, the usual products of inflammation were found—redness and vascularity, lymphy exudations, purulent serum with albuminous flakes. To take a typical example of the intra-peritoneal form from M. Voisin:—On a level with the brim of the pelvis, the viscera were seen to be united together, forming the roof of the cyst. The bladder was elevated above the pubes; the uterus close behind it, somewhat increased in size, and rotated upon its axis into a position different from the usual one. Behind, adhesions united the posterior and superior aspect of the uterus to the rectum, a portion of the sigmoid flexure of the colon and several coils of small intestine, the two broad ligaments and the posterior half of the circumference of the brim of the pelvis. A roof was thus formed over the posterior half of the pelvic excavation. On laying open the cyst, the thickness of the walls was found to vary with the amount of fibrinous exudation at the point of incision. The cyst cavity was divided into a number of compartments by cellular bands, but communications existed between the various loculi. All the pelvic organs were more or less fixed, the ovaries displaced, and completely lost among the inflammatory products. In an opening that had been effected previous to the decease of the patient, traces of ulceration were found, and the fistula between the aperture and cyst was sinuous and irregular.

The contents of the cavity sometimes consist of clots more or less changed in colour and arrangement; sometimes of a variable quantity of black fluid, greyish at certain points, and sometimes like an admixture of soot and water. At times the fluid has a tarry, syrupy consistence, and if suppurative action has taken place, there is an admixture of pus. These products have been observed also when the cyst has been evacuated during life by a trocar. Under the microscope the contents have been found to be composed of blood globules completely bereft of colour, and so altered in shape as to be scarcely recognisable. Besides these are fat globules, amorphous particles of hæmatoidine, various crystals, and other materials resulting from the transformation of effused blood. In most cases of encysted hæmatocele, the displacement and confusion of parts seems to have been so great in consequence of the effused blood and subsequent inflammation, that the determination of the source of hæmorrhage was anything but satisfactory. From various data, however, the blood seems to have come from rupture in an ovary previously diseased, in the largest number of instances.

Causes.—The causes are predisposing and exciting. As might be expected, hæmatocele occurs at the period of greatest sexual vigour in women; and from an extended series of observations, collected by Dr. Tuckwell, it has been found that between twenty and thirty years of age is the period of its most frequent occurrence. Marriage seems to have little influence in its production. Some deviation from normal conditions in the function of menstruation has been noted by all observers, as preceding the development of hæmatocele. Thus M. Voisin remarked that the larger number of patients affected with hæmatocele had habitually an over-abundant menstrual flow; the colour of the discharge was bright, and clots were frequent. These signs he considers as indicating a plethoric condition of the system, and infers that a recurring over-distension of the pelvic blood-vessels in plethoric patients favours the formation of hæmatocele. On the other hand, hæmatocele undoubtedly occurs from time to time in feeble and chlorotic patients, whose menstruation has been suspended it may be for months, and who are the subjects of amenorrhœa. In these cases, the rupture of an internal blood-vessel does not necessarily take place from any physical obstruction to the catamenial flow by the natural passages, but from constitutional conditions which have impaired the quality of the blood and weakened the integrity of its containing vessels.

Pathologists are familiar with attacks of hæmorrhage from mucous canals, and into serous cavities, during the progress of eruptive fevers, in purpura and other cachectic conditions, and similar morbid changes in the blood seem to be predisposing causes of hæmatocele.

In most patients whose menstrual history previous to an attack has been investigated, it has been ascertained that the recurrence of the periods was regular, and the discharge was frequently profuse and too prolonged, but whether abundant or scanty, it was in the majority of cases attended with pain. In a few of these the pain may have been due to mechanical obstruction, caused by a contracted cervix or a displaced fundus, but in most instances the suffering was but the expression of faulty performance of function on the part of the womb, associated with an over-distension of its blood-vessels. Among the other predisposing causes, a weak and varicose condition of the veins in the pelvis and lower extremities deserves a place. Women who have varicose veins of the lower limbs, and are liable to hæmorrhoids, to venous swelling of the vulva, and to a weighty, spongy condition of the uterus, often menstruate most copiously and painfully. These patients have frequently been observed to be prone to hæmatocele, when an exciting cause has been applied during or near the time of a menstrual period.

The exciting causes enumerated are, sudden suppression of the catamenial flow, over fatigue, violent straining at stool, cold, intense mental emotion, premature exertion after abortion, and violence producing injury during menstruation. In a considerable number of Voisin's cases the immediate cause was traced to coitus which had taken place either during the catamenial flow or very shortly after its termination, and the pain began during the sexual act.

SYMPTOMS AND PROGRESS.—The symptoms of non-encysted extravasations and of cystic hæmatoceles differ in some important particulars. In the former, the commencement of the disease is sudden, perhaps coming on in the midst of apparently good health, and the suddenness and intensity of the attack, it has been suggested, may possibly lead to a suspicion of poisoning. It is commonly coincident with a menstrual period. The initiatory symptoms are in all cases those of severe shock with intense abdominal pain, similar to that produced by an attack of acute peritonitis, the patient being thrown into the most violent distress and agitation. The symptoms indeed bear a very close resemblance to those produced by perforation of the stomach or bowel, with extravasation of their contents into the peritoneum; but in addition there is marked anæmia produced by sudden and profuse loss of blood. The belly becomes tender and hard, as well as dull on percussion. Hiccup and vomiting are sometimes present; the temperature of the skin is low, and its surface pale and blanched. Syncope or complete collapse speedily follows, with a small and almost imperceptible pulse, and death generally ensues in less than twelve hours.

The incipient symptoms of encysted hæmatocele are to some extent the same in character as in the non-encysted, but those common to both are somewhat less in severity. The gravity of the attack varies in accordance with the suddenness and amount of blood extravasated, and the general condition of the patient. It is highly probable also that the severity of the attack will vary with the seat of the effused blood, being more acute and threatening when the blood is poured into the peritoneal cavity, less so when the effusion is into the cellular tissue, for the double reason that less disturbance is provoked when blood is extravasated beneath the peritoneum than on its free surface, and that effusion is likely to be slower and more gradual into the cellular tissue.

The illness, as in the other form, is generally preceded by some notable derangement in the menstrual function, and commonly dates from a catamenial period, which has perhaps been attended with more than usual pain; the discharge being inordinately profuse, and prolonged beyond the normal limits. Then immediately following some effort, or coitus, comes sudden and intense pain in the pelvis, often compared to the throes of parturition, but increased by the least pressure or movement. If the blood effused be considerable in quantity, and particularly if it has escaped into the peritoneum, there is fainting almost amounting to syncope, and this conjoined with the indications of local peritonitis. In several instances it has been noticed that the patient, having been exposed to cold or undue exertion or injury during menstruation, or immediately after it, has awoke in the night with a sense of exhaustion and faintness, and begged to be supplied with food. This preliminary exhaustion has speedily been succeeded by abdominal pain and other characteristic symptoms. The pain is more or less continuous with recurring exacerbations, or it is paroxysmal, and a weight about the anus is often complained of, with frequent desire to go to stool. Pain and difficulty are frequently experienced in evacuating the bowels, with tenesmus, and quantities of mucus passed, possibly mixed with blood, indicate much irritation of the intestinal mucous membrane. Painful micturition is not uncommonly complained of, and partial retention of urine may greatly add to the discomfort of the patient, while, if not detected, it may lead to complications in diagnosis and mask the real ailment. To these symptoms Voisin adds, a rapidly produced and marked pallor of the skin, which assumes a dull whiteness, and is not unlike that which accompanies the cancerous cachexia. Very speedily acute febrile symptoms supervene with rapid pulse, increased temperature, and loaded urine. The patient prefers to lie upon her back with the thighs flexed on the abdomen. There is often considerable distension of the intestines by flatus, and all the ordinary signs of limited peritonitis are developed as the result of the sanguineous effusion.

If the hæmatocele be large, a tumour is speedily discovered which can be felt through the abdominal walls above the pubes, or in the direction of the iliac fossa on either side. Dr. West says he has detected the swelling within 48 hours after the first symptom, and in many cases it may probably be detected earlier. It is commonly only slightly tender to pressure, but in some instances careful examination is rendered impossible for a time by the extreme sensitiveness. It will be best examined as the patient lies upon her back, because then external and internal manipulation can be combined, and the most accurate estimate formed of the size, consistence and relations of the tumour. A careful exploration by the rectum should rarely be omitted, as in this way the position of the swelling as it lies between the vagina and bowel is at once ascertained.

The physical characters of the tumour are dulness on percussion, immobility, or very partial mobility, more or less irregularity of outline, and generally some tenderness on pressure. Soon after its formation, the swelling is elastic and indistinctly fluctuating; later, it is irregular and of unequal density, and then closely simulates the results of pelvic cellulitis. When of any considerable size, and retro-uterine, it is found on vaginal examination to occupy the posterior half or more of the true pelvis, elevating and pushing forward the cervix uteri above the pubes, stretching and distending the posterior wall of the vagina, and compressing the rectum behind it, into the concavity of the sacrum.

Ordinarily, it seems fused with and firmly united to the uterus, but occasionally the uterus may be moved in some degree independently of the swelling, both with the finger and the uterine sound. Dr. M. Duncan noticed that the length of the uterine cavity was much increased whenever the hæmatocele was large, and it decreased with the contraction of the blood sac. Frequently it has been observed of such dimensions as almost to fill the true pelvis, and to distend and push down the back wall of the vagina so far that it almost reached the vulva. When the swelling projects very low in the pelvis, it has been supposed that it must necessarily be due to extravasation into the cellular tissue, because the peritoneal sac has a higher level; but when it is recollected that the peritoneum is often prolonged far down the posterior wall of the vagina, and that the lower boundary of the *cul de sac* almost reaches to the floor of the pelvis, this deduction is found to be of uncertain value. A source of fallacy concerning the very low position of the tumour exists in some cases of hæmatocele, as well as in pelvic cellulitis. This consists in the production of a large amount of œdema in the lower part of the recto-vaginal septum. This is sometimes so considerable as to form a distinct rounded swelling within the vagina. When the position of the tumour is other than retro-uterine, it will displace the pelvic organs in accordance with its dimensions and relative position. On more than one occasion it has been remarked that it produced complete retroversion of the uterus. Graily Hewitt, in his "Diseases of Women," gives two illustrations of position, in outline, of the extra-peritoneal form. In one, the hæmatocele tumour rose as high as the crest of the ilium on the right side, and dipped half way down the pelvic canal inferiorly. In a second the extravasated blood is represented as surrounding the bladder, uterus, and rectum in every direction, and the tumour so formed reaches as high as the umbilicus above, and to within a short distance of the perineum below. These, however, are extreme forms. Occasionally the quantity of blood effused is so limited that no well-defined tumour is formed notwithstanding the presence of characteristic, general symptoms. Dr. West, who with Dr. Matthews Duncan has noticed the absence of distinct tumour in some cases where the other symptoms were unequivocal, believes that in these instances the effusion was extensive, and either not circumscribed enough to form a defined tumour, or at any rate not low enough in the pelvis to produce bulging of the vagina.

This explanation obviously will not apply to those cases in which the general symptoms, though the same in kind, are slight in degree, and it seems more reasonable to suppose that the effusion has been so scanty as to produce but little local tumefaction.

It has been observed, but rarely, that more than one hæmatic tumour was present at the same time; one being seated in the iliac region and felt by external examination, the other being situated deeply in the pelvis and reached only by vaginal exploration. It is of course possible that these apparently separate tumours may have been the extreme poles of one elongated tumour. Some authors have enumerated among the symptoms an undue pulsation of the arteries in the vagina and cervix uteri as observable during the progress of hæmatocele, but this symptom is not always present, and in Dr. Madge's case,[1] both internal throbbing and the pain in defecation previously mentioned were notably absent.

The progress of the disease is variable, and so also is the development of the tumour. Sometimes the attack sets in with great violence, and the progress is rapid. In the majority of cases, indeed, the tumour attains its largest size in a very short time from the commencement. In a few hours, or at most, a few days after the first symptoms, the pelvic mass may attain the size of a child's head, or of a gravid uterus at six months, and it then rarely increases in size afterwards. Other instances present themselves in which the symptoms are less acute. Blood seems to be poured out in but small quantity and in progressive quantities at certain intervals, creeping on, as it were—the increase in swelling coinciding with the monthly periods. Nevertheless a careful study of well-marked cases has shown that very speedily after the swelling has reached its maximum development, the natural tendency is to decrease in size. The tumour gradually becomes harder to the touch, of unequal density, and the sensation of fluctuation gradually disappears. These alterations in consistence arise from the changes which take place in the extravasated fluid. The serum of the blood is quickly absorbed, while the coagulum remains for a much longer period, some portion of it being found occasionally even months after the date of the attack. When the hæmatocele has attained its maximum size and become stationary, it has been noted by several writers as a curious fact that the recurrence of the menstrual period exercises no influence in increasing its dimensions. On the contrary, when menstruation is re-established, each period is marked by a notable diminution in volume, and instead of undergoing a gradual and continuous decrease, the swelling recedes by successive steps, which correspond to catamenial periods. This fact is especially dwelt upon by M. Voisin and Professor Dolbeau. With the invasion of the malady the catamenial flow, if present at the time, may be checked and only return after an uncertain interval. Very commonly, however, instead of being arrested, it becomes so profuse as to be a marked feature in the case, and when restrained within moderate limits, it often persists for weeks as a dribbling hæmorrhage, which is a further drain on the strength of the patient.

After a time, in the natural course of events, when no surgical procedure is undertaken, and judicious palliative measures have been adopted, the febrile symptoms gradually subside, the pain decreases, and subsequently the patient experiences only great weakness; a sense of weight in the pelvis, bearing down; some difficulty in micturition and defecation; and pain and discomfort in attempting to walk or to assume the sitting posture. If one side of the pelvis only is occupied by the tumour, there may be increased pain on moving the leg of the affected side. These symptoms are variable, but as a rule when no interference is practised, recovery takes place slowly and by resolution, the blood being gradually absorbed and the damage done being ultimately repaired. The average period, within which cure takes place in this way, is found to be about four months. Small collections of blood may under favourable circumstances be removed by absorption in six weeks; while if the tumour be large, or recovery has been interrupted by any cause, eight, twelve, or more months may elapse before all traces of induration in the pelvis have disappeared. When recovery does not take place by resolution, the fluid contained in the hæmatocele commonly makes its escape externally through one of the pelvic canals. There is a divergence of opinion as to whether suppuration of the cyst always precedes its evacuation, but the failure to detect well-formed pus in any considerable quantity in the discharges, in most of the instances which have terminated in this way, evidently points to the conclusion that there has been no general suppuration of the cavity, but that simple ulceration of the walls takes place resulting in perforation and subsequent evacuation. When such evacuation does take place, a reaccession of the original symptoms commonly occurs, and is then followed by the discharge of a quantity of fluid and semi-solid material which has been compared in appearance to currant jelly, and in odour to that of decaying flowers. In twenty-seven instances cited by Voisin, six emptied themselves by the rectum, three by the vagina, and four burst into the cavity of the peritoneum. This last mode of termination, whether produced by suppuration or not, is by far the most serious, inasmuch as it is uniformly followed by general peritonitis and death. If suppuration should take place in the hæmatic cyst, it is generally associated with a fresh accession of pain—rigors and fever, followed by profuse perspirations, as when suppuration is established in other parts. The danger of a rupture into the peritoneal cavity is then considerably increased, and hence the necessity of early artificial opening when once suppuration is established beyond doubt, and the symptoms are threatening. Dr. M. Duncan has dwelt on the importance of recognising the existence of fluid in the lowest part of the sac in hæmatocele of some standing, as indicative of previous inflammatory action and the presence of pus. More recently he has modified this opinion, and has pointed out that the mere presence of fluctuation, if it has not been preceded by the general and local signs of suppuration, is not sufficiently trustworthy, and may lead to an erroneous conclusion. The inflammation of a hæmatocele, particularly if it suppurates in such a way and points in such direction that it cannot readily be opened by the knife, is apt to protract the recovery of the patient indefinitely. At times the formation of purulent matter takes place so insidiously that the signs are too indefinite to act upon, and the first distinct proof of its existence is the discharge of pus and broken-down coagula or coffee-ground materials by the rectum. As in pelvic abscess, this may lead to chronic irritative diarrhœa and dysentery, the prolongation of which, added to the profuse purulent discharge and accompanying hectic, may exhaust the strength of the patient and lead to a fatal result. An example of this kind is given by Dr. McClintock in his "Diseases of Women." In Dr. Madge's case (Obstet. Trans.), in addition to suppuration and exhausting discharges, phlegmasia dolens set in as a further complication, and the patient died from extreme exhaustion.

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[1] Obst. Trans., vol. 3.

Lastly, it may be mentioned that intercurrent peritonitis may at any time supervene during the progress of the affection, and this apparently apart from rupture of the cyst. Commonly, the inflammation of the peritoneum which is lighted up at first in all cases when blood is poured into the peritoneal sac, gradually subsides after the effusion is sealed up by surrounding adhesions, and in these instances the patient has a fair prospect of recovery. If the peritonitis becomes more general, this entails further peril, and at no time during the continuance of the hæmatocele is there immunity from its possible reappearance. Voisin has observed this mode of fatal termination in one case as late as three months, and in another at the end of four months from the production of the hæmatocele.

DIAGNOSIS.—In a typical case of pelvic hæmatocele the diagnosis is commonly made without difficulty. The occurrence of the attack at or about the date of a catamenial period, the menstrual function having previously been notably deranged; the suddenness of the invasion and the production of general anæmia; the rapid formation of the pelvic tumour, accompanied first with symptoms of shock, and later with those of limited peritonitis, are the points chiefly to be dwelt upon as the cardinal elements in forming an opinion; and if the case has been under observation from the commencement, the real nature of the affection is not likely to be overlooked. When, however, a case is seen for the first time in its chronic stage, the formation of a correct opinion is often most difficult; and even with all the aid of a succinct history, which is not always forthcoming, it may readily be mistaken for other morbid conditions found in the pelvis.

1. The affection which of all others bears the closest resemblance to pelvic hæmatocele in its chronic stages, and which is most likely to be mistaken for it, is, peri-uterine inflammation, pelvic cellulitis, or peritonitis, or pelvic abscess, as it has been variously named by different authors. The inflammatory affection is more frequent than hæmatocele, but so many symptoms are common to both, that the greatest care is often needed to form a correct diagnosis, and in some cases it is doubtful if the nicest discrimination will prevent the one from being mistaken for the other. The chief points of difference in the history of the two affections are that pelvic inflammation and abscess are more frequently consecutive to abortion and delivery, and when not so, have

often some relation to inflammation previously existing in the uterus or ovaries. Hæmatocele, again, is more constantly connected with some accidents of menstruation. Pelvic cellulitis or peritonitis has no such common coincident menorrhagia, and does not develop its greatest intensity suddenly; the skin does not rapidly become pale from anæmia, as in the larger hæmatic tumours; and the swelling, if watched throughout its course, is more likely to begin in the lateral and anterior parts of the pelvis; it is hard at first, comparatively slow in formation, and fixes the uterus to the walls of the pelvis; later, if suppuration occurs, it then becomes soft and fluctuating. In hæmatocele, on the other hand, the tumefaction is more frequently behind the uterus, it is soft in its early stages, and grows harder as time passes, only beginning to fluctuate again if the cyst becomes inflamed and suppurates. A further point of difference to be noted is that in hæmatocele, if the swelling be at all considerable, it has a tendency to roundness in form, with hard inflammatory deposits about its margin; and it displaces and distorts the uterus from its natural position. In cellulitis and pelvi-peritonitis the fibrinous deposit is infiltrated through the pelvic tissues affected, fixing the uterus so that it cannot be elevated or depressed, instead of displacing it. It often gives the impression which might be produced by soft plaster of Paris poured into the pelvis and hardened there, fixing the viscera to the osseous boundaries of the pelvis. The constitutional symptoms also follow an inverse ratio in the two affections, febrile disturbance distinctly *preceding* the formation of tumour in the inflammatory affection, *following* its formation in hæmatocele.

When the amount of extravasated blood is small, it may be impossible, either in the early or later stages, to discriminate between the slight thickening and induration produced by this cause in the pelvic tissues and that arising from primary inflammation. Equally when a case does not come under observation until suppuration has occurred, and no distinct history can be elicited, it may be impossible to determine whether the abscess is due to primary phlegmonous inflammation or is the secondary product of a suppurating hæmatocele. Fortunately in the two cases the treatment is practically the same, and the patient suffers no disadvantage from a failure to decide concerning these perplexing differences.

2. The diagnosis between hæmatocele and ovaritis is stated by M. Voisin and others to be very difficult, and yet one would fancy that practitioners familiar with the varying phases of inflammation of the ovary were not likely to commit an error of diagnosis between the two. The limitation of the pain and swelling in the earlier stages to the locality of one or other ovary, with the general symptoms, would be a sufficient guide to the nature of the affection in the larger number of cases. When a considerable swelling has formed as a consequence of a longer and more intense attack of ovaritis which has extended to surrounding parts, it may be needful to remark that there has been no sudden invasion or rapid formation of a tumour, as in

hæmatocele; no sudden anæmia, perhaps no coincident menorrhagia; and the symptoms gradually increase in severity from the commencement, while in hæmatocele the most severe and distressing symptoms are developed from the first, and as time passes, undergo gradual amelioration.

3. The various forms of ovarian tumour of limited size are in general to be distinguished by their lateral position, slower growth, mobility, circumscribed form, and absence of urgent symptoms from the commencement. If an ovarian cyst, while small, has descended into the retro-uterine *cul de sac* and formed adhesions there, the points of difference indicated become less useful guides, and if perchance the cyst inflames and suppurates in this position, the diagnosis may be by no means easy. Ovarian tumours indeed sometimes inflame while lying behind the uterus, and without proceeding to suppuration throw out irregular lymphy deposits which mask the circumscribed form of the original tumour, and with softened centre and harder circumference may simulate the physical characters of hæmatocele. The only way of discriminating in these perplexing cases is to study carefully the previous history and watch the further progress. It is useful to bear in mind, as pointed out by Dr. Mc Clintock, that it is extremely rare for an ovarian cyst to sink as low in the pouch of Douglas as the blood gravitating downwards in the peritoneal cavity, when an intra-peritoneal hæmatocele is formed. This indication, however, loses much of its value from the fact that when inflammation of any kind occurs in the neighbourhood of the pouch of Douglas, there is often so much œdema of the recto-vaginal septum that it closely resembles fluctuation there, and often forms a considerable projection of the posterior wall of the vagina. In the lapse of time, after the inflammatory action has subsided, the nature of the disease is made clear by observing that, as the serous and lymphy effusion is absorbed, the central tumour remains, and if this be fluctuating and unattended by hectic symptoms, it is pretty certain to be ovarian. In doubtful cases of this kind, where it is of importance to decide speedily the character of a fluctuating swelling in this locality, recourse may be had to an exploring needle or fine trocar, as recommended by Sir. Jas. Simpson and Professor Braun.

A very difficult point in diagnosis may arise, between the formation of hæmatocele and the sudden and rapid enlargement of an ovarian cyst by hæmorrhage into its cavity. In both there would be the general indications of internal hæmorrhage, but in the latter case there would probably be the history of a pre-existing ovarian tumour, and the perfect regularity and smoothness of the swelling, with its possible mobility, would favour a correct inference as to its character.

Encephaloid tumours, which, from their consistence and position, may be mistaken for hæmatocele, are to be distinguished by their history and the presence or absence of the cancerous cachexia. It is important, however, to bear in mind that the appearance of a patient suffering from an extensive hæmatocele is very like that of a cancerous patient, and if the aspect and colour of the patient be too implicitly relied upon, it may lead to serious error.

4. Retroflexion and retroversion of the uterus in the gravid and unimpregnated conditions,—inasmuch as they may be produced suddenly from violent efforts or straining, and lead sometimes to retention of urine and other urgent symptoms coincident with the formation of a retro-uterine tumour,—may be mistaken for hæmatocele. In the unimpregnated condition, the use of the uterine sound, showing the direction of the womb cavity, would at once solve the question. When menstruation has been suspended and there are other signs of pregnancy, the sound cannot of course be employed, but there will ordinarily be no great difficulty in arriving at a correct conclusion if the general course of the symptoms be carefully studied, and if due care be exercised in observing the circumscribed form of the swelling, the absence of adhesions on its borders; its relation to the cervix uteri, and its partial mobility and in some cases perhaps its capability of being replaced by pressure with the finger through the vagina or rectum. The value of any signs connected with the mobility of the swelling is necessarily vitiated, both in the pregnant and non-gravid condition, when the fundus is bound in a retroflexed position by old adhesions. Wherever the hæmatocele is of any considerable size it may be felt above the pubes, while a retroflexion can only be reached by internal examination.

5. Fibroid tumours of the uterus are to be diagnosticated by their history of slow, commonly painless growth, by their density, and relations, and their attachments to the uterus. In their commonest forms they bear no sort of resemblance to hæmatocele either in their mode of development or physical characters, and yet the disastrous experience of such experts as M. Malgaigne and Professor Stoltz plainly shows that some of the complicated forms of fibroid may be mistaken for hæmatic tumour. As mentioned in the earlier pages of this memoir, M. Malgaigne proceeded to remove by operation a supposed fibroid tumour of the uterus which proved to be a retro-uterine hæmatocele, and Stoltz fell into the same error, both cases ending fatally. In truth, the difficulty of determining in some cases whether a pelvic tumour is solid or has fluid contents is almost insurmountable without the aid of an exploring needle. A tightly distended cyst may feel so hard and dense as to simulate a solid growth, and on the other hand a fibroid tumour may possess such softness in its component elements, particularly if previously inflamed, that it is impossible to distinguish by the unaided touch between the two. When it is added that both fibroid tumour and hæmatocele are commonly attended by menorrhagia, which makes another point of resemblance, it will be obvious that no serious operation should be undertaken in this class of cases without previously clearing up any doubts which remain, by the exploring needle.

6. Fæcal accumulations in the lower bowel are to be distinguished from hæmatocele by their plastic character, *i.e.* they may ordinarily be

indented by pressure through the vaginal wall. If harder, a careful digital exploration by the rectum will reveal the true nature of the swelling.

7. Lastly, some of the forms of extra-uterine gestation bear so close an analogy to pelvic hæmatocele, or general blood extravasation into the peritoneum, that even the most experienced observer may be unable to decide between the two. MM. Robert and Huguier mistook a tumour, which turned out to be an extra-uterine fœtation and occupied a considerable space in the pelvis and abdomen, for a pelvic hæmatocele. The absence of urgent symptoms in the commencement, and the presence of the usual signs of pregnancy, are the chief guides in discriminating between the two conditions. A still greater difficulty presents itself, however, when an early extra-uterine fœtation bursts into the cavity of the peritoneum, as ordinarily takes place when the ovum is developed in the Fallopian tube. In these instances the actual symptoms are absolutely the same as in intra-peritoneal pelvic hæmatocele, and the same pathological result is brought about, viz., the sudden escape of a quantity of blood into the peritoneum. Unfortunately the abundance of the sanguineous effusion in these cases gives no time or opportunity for its limitation by adhesions, and commonly the patient speedily dies from shock or peritonitis. There is nevertheless reason to believe that death does not always ensue in these instances, but that the extravasated blood and ovum become surrounded occasionally by adhesions after the manner of encysted hæmatoceles, and thus recovery takes place. In all cases it may assist in the formation of a correct appreciation of the facts, to recollect that even in extra-uterine pregnancies there is ordinarily suspension of the catamenia for one or more periods, symptomatic changes in the mammæ, alterations in the cervix and body of the uterus, and sympathetic disturbance in other organs. Possibly, before the sudden invasion of the illness, a swelling may have been detected in process of development on the lateral margin of the uterus.

The PROGNOSIS is always more or less an anxious one. When vascular rupture has taken place into the pelvis, it is impossible to foresee to what extent the hæmorrhage may extend, or when once checked what amount of exertion and how slight a movement may bring a renewal of such extravasation as may prove fatal. The tendency to aggravation at the catamenial periods, and the liability to low forms of peritonitis creeping on in patients apparently not in immediate peril, or even where the cyst is contracting and convalescence seems progressing, are additional sources of anxiety. To these may be added the feebleness and exhaustion sometimes produced when the cyst suppurates and discharges through the bowel or vagina, and the not infrequent supervention of general blood-poisoning from absorption of morbific materials into the general system. With these detractions, the prognosis may as a rule be stated to be favourable. In the larger number of cases hæmatocele is not fatal, and by judicious management an untoward result may often be avoided, even when the symptoms are severe. Dr. F. Weber, of Berlin, one of the most recent writers on the subject, considers the prognosis very favourable. Of twenty-three cases observed by him, none were fatal—a result he ascribes to his method of treatment,—ice bladder,—perchloride of iron internally, and *avoidance of puncture.* In three of his cases, the broken-up extravasation burst through the rectum.

The TREATMENT may be considered as prophylactic and therapeutic.

Although it may be impossible to foresee an attack of internal hæmorrhage forming hæmatocele, yet since patients suffering from certain derangements or forms of weakness are found by experience to be the most frequent subjects of hæmatocele, they should, when possible, be guarded by special precautions. Thus, women who suffer from dysmenorrhœa, particularly the obstructive forms, where mechanical hindrance exists to the escape of the catamenial discharge, constitute a large proportion of those affected, and these patients should be warned to be at rest during the continuance of the menstrual period, and in the intervals be submitted, if practicable, to such treatment as will remedy the painful and difficult menstruation. Again, women with varicose veins of the lower extremities, of the vulva or rectum, and who menstruate very profusely as well as painfully, should especially avoid all the exciting causes which are known to provoke hæmatocele. Such persons should avoid travelling, violent exertion, or exposure to cold, during the continuance of the catamenia, and sexual intercourse should be interdicted altogether near the menstrual period, and should at all times be practised moderately and without violence.

The therapeutic management must necessarily be modified by the *stage* of the affection which the medical man is called upon to treat. In the stage of shock or depression resulting from the first sudden blow inflicted by the effusion of blood, it is needful to bear in mind the pathological cause, and while palliating the general symptoms of shock, to try and limit the extravasation as much as possible. Non-encysted extravasations are as a rule so speedily and certainly fatal, that probably little can be done to avert a fatal issue; as however there are no means of accurately ascertaining the extent of the effusion, and consequently of judging of the probability of its becoming encysted, it behoves the practitioner when the first symptoms of internal hæmorrhage present themselves, to adopt such measures as are likely to stay the flow of blood, promote its coagulation, and avert the more disastrous consequences. The indications therefore are *not* to apply hot poultices or fomentations to relieve the abdominal pain, but cold compresses or bladders of pounded ice over the hypogastrium, to arrest internal hæmorrhage. The patient should keep the horizontal posture absolutely, and avoid every movement of the body and all mental emotion which may disturb or excite the general circulation. Further aid may also be afforded by the use of sinapisms to the upper extremities, and a full dose of opium will have the double effect of calming the pain and lessening the depressing effects of the loss of blood. Some French authors recommend that in this stage blood should be taken once or twice from the arm to produce a derivative effect on the pelvic vessels and depress the systemic circulation, thus lessening the pressure on the internal bleeding points. M. Aran, in cases where the constitutional condition of the patient will permit, applies twenty to thirty leeches over the abdomen on the first day; fifteen to twenty on the second, and twelve to fifteen on the third day, if feebleness is not too great. The strength is supported by nutritious diet during the depletion, and the leeching is followed up by blisters and counter irritation to the abdomen as soon as possible. M. Aran speaks strongly concerning the favourable issue and short duration of cases treated by this method. Neither general nor local depletion at this stage have, however, met with much favour in Great Britain. The diet should be simple, unstimulating, only enough to prevent the exhaustion of the patient, and all drinks should be taken cool or cold. Brandy or other stimulants should only be given when there is great prostration or collapse, lest they quicken the circulation and thus favour a larger loss of blood. Various astringents and hæmostatics may be administered if this be thought desirable. Sulphuric acid, tannin or gallic acid, acetate of lead, or perchloride of iron, may be combined with opium. When the alarming symptoms of the first stage or shock have subsided, it may be needful to calm the feverish reaction which commonly follows, and to combat the indications of local peritonitis which remain. To further this object, repeated doses of opium or some other sedative and narcotic should be administered by the mouth or rectum; the patient should be absolutely at rest, the bowels kept quiet, and in the more critical cases the urine should be drawn off with a catheter, rather than allow the patient to make the least exertion. If the signs of local peritonitis are very severe, and the patient's strength will bear it, the application of a few leeches to the hypogastrium or iliac region may be recommended with advantage. Even in this stage, warm local applications should be used with caution, as they are apt to promote a fresh access of hæmorrhage, especially in the intra-peritoneal form, and favour the breaking down of the yet young lymph adhesions.

Opinions differ considerably concerning the propriety of surgical interference after the more acute symptoms have passed. Some authorities recommend puncture and evacuation in those instances where a distinct tumour is formed, which projects prominently down into the pelvis, and gives indications more or less distinct of fluctuation. Others counsel non-interference; and others again pursue an eclectic method and prefer evacuation by surgical means only when the symptoms are urgent enough to call for it.

It appears that at one time M. Nélaton employed the method of puncture and evacuation in all cases indiscriminately. In several instances where puncture was practised, his patients were attacked with purulent infection and died. This led to a modification of treatment, and artificial evacuation was resorted to only when the symptoms were urgent. According to M. Nélaton's later teaching, surgical interference is only warrantable when such symptoms are present as lead the practitioner to fear that rupture of the adhesions forming the parietes of the cyst will take place, and lead to subsequent extravasation of its contents into the general peritoneal cavity. Thus, where a hæmatocele of considerable size already exists, and appears to be increasing in size, being attended by constant and violent pain, he concludes that secondary inflammation is going on in the cavity, and that unless an opening be effected to lessen the tension, there is every probability that laceration of the cyst-walls will take place, and a general and fatal peritonitis be the result. M. Nélaton would not operate merely to expedite the recovery of the patient if there were no threatenings of immediate peril. The statistics collected by M. Voisin and published in his monograph are decidedly adverse to the propriety of artificial evacuation as a general plan of treatment, and lead him to prefer an expectant method, unless the case is attended with exceptional and threatening symptoms. Thus out of 20 cases where surgical interference was resorted to, 15 recovered and 5 died. In contrast with this, out of 27 cases treated by the expectant method, 22 recovered and 5 died. Deducting from the last class 2 deaths, in which hæmatocele was apparently not the immediate cause of death, the mortality was reduced to 1 in 9 when no operation was performed, while in cases where an artificial opening was made, the mortality was 1 in 4. Great as this difference may seem, it cannot be taken as overwhelming evidence in favour of leaving all cases indiscriminately to the curative powers of nature. M. Voisin's statistics are too limited for founding a sound practical conclusion, and there are sources of fallacy in them which do not lie immediately on the surface. The force of these objections is illustrated by comparing the results of a large number of cases tabulated by Dr. West in his Lecture on Hæmatocele. Of 55 cases treated on the expectant plan, 43 recovered and 12 died; while of 48 cases in which surgical interference was had recourse to, 40 recovered and 8 died. It deserves, moreover, to be pointed out as probable, that some of M. Voisin's cases treated by puncture, and which terminated fatally, were instances of the worst form of the disease, and that an opening was deemed necessary on account of the severity of the symptoms, not because it was deemed the best method as a general plan of treatment.

Recently, Dr. Meadows has published a paper in the thirteenth volume of the "Obstetrical Transactions," in which he boldly advocates the desirableness of more frequently resorting to puncture when the quantity of effused blood is more or less considerable. He supports his advocacy by an appeal to the statistics of M. Bernutz, although that author himself is strongly opposed to the practice in question. Abstracting from M. Bernutz's cases such instances as he thinks may fairly be taken as illustrating the advantages and disadvantages of evacuation, he points out that of 19 cases in which evacuation took place either by spontaneous rupture or by tapping, 16 recovered and only 3 died; while of 11 cases in which no evacuation occurred, 2 only recovered and 9 died. Statistics concerning the value of methods of treatment are avowedly untrustworthy, because it is difficult to eliminate sources of fallacy. Notwithstanding, therefore, the figures quoted above, it may be broadly stated that the weight of opinion is decidedly adverse to artificial evacuation of the contents of hæmatocele as a general method of treatment, and that only in exceptional cases when the symptoms are urgent is an operation desirable. The sources of danger attending artificial evacuation are, purulent infection, provoked in some degree probably by the admission of air into the sac, and the putrefaction thus set up; the recurrence of secondary peritoneal inflammation, and the renewal of the hæmorrhage from its original sources when the pressure of distension is removed. A patient under the conjoint care of MM. Malgaigne and Nélaton died from hæmorrhage of a posterior uterine artery which was wounded by the puncture; and a patient operated upon by M. Huguier died of peritonitis provoked by injecting warm water to wash out the contents of the cyst.

The results of some recent experiences, nevertheless, testify that puncture of hæmatocele carefully conducted is somewhat less perilous than was at one time supposed, and hence the operation may be the more readily undertaken in appropriate cases. Thus Dr. Matthews Duncan, a very careful observer, has less fear of untoward results following than formerly prevailed, and he believes that he has repeatedly shortened the duration of the malady by artificial opening. Professor Braun also has observed the rapid cure following the emptying of hæmatocele by puncture.

After a careful examination of all the circumstances, it may be stated in summary that as a general plan of treatment it is most prudent to adopt a palliative method, relieving the symptoms by appropriate remedies as they present themselves, and taking such precautions as are likely to ward off fresh complications. This palliative method should be pursued irrespective both of the size and position of the hæmatocele, and in a large proportion of cases, if perfect quiescence can be enforced, the tumour, even if of considerable dimensions, will gradually and slowly disappear. If, however, all precautions prove fruitless, and the tumour instead of subsiding, remains long of the same size, or rather shows a tendency to increase, and if, besides, there are constant and severe pains, the recurrence of rigors and marked increase of temperature at nights, with other hectic symptoms, which indicate that suppuration has taken place, then artificial evacuation becomes justifiable and necessary. In some cases, where no distinct signs of suppuration have taken place, the urgency of the symptoms may yet call for operative interference. Thus the persistence of severe and exhausting chronic vomiting, which has been observed in one or two cases associated with large hæmatoceles, and continued and alarming obstruction of the bowels notwithstanding the use of appropriatere medies for its relief—as in a case described by Dr. Meadows—may urgently call for some diminution in the amount of physical pressure only to be obtained by puncture. In undertaking the evacuation of the cyst, great care must be taken not to operate so long as there is any reason to suppose that hæmorrhage is still going on within; and further, a sufficiently long time should be allowed to elapse after the formation of the tumour and after the latest increase to its bulk, to permit its boundaries to become consolidated. A too early opening in the intraperitoneal form would most probably lead to the breaking down of the recently formed adhesions constituting the cyst-wall, and the lighting up of fresh and perhaps fatal peritonitis.

In selecting the locality for puncture, the vagina should always be preferred if evacuation there be possible, as the cyst-contents thus gravitate readily from the opening, and less irritation is provoked than when evacuation takes place through the bowel. The irritating and exhausting diarrhœa produced either by spontaneous or artificial evacuation into the rectum frequently adds a new source of danger to a patient already much enfeebled by the previous progress of the affection, and it is well to avoid this if a choice is permitted. In a case of Dr. West's, puncture by the vagina did not prevent a spontaneous opening through the bowel, and hence, if spontaneous evacuation by the rectum seems inevitable from the pointing in that direction, it may be best, notwithstanding the disadvantages named, to expedite escape in that direction, rather than run the risk of a double opening. When the swelling is not readily accessible by the vagina, but reaches high up into the abdomen, it may be possible to puncture it through the abdominal walls; and if there be any obvious pointing externally, it is safer to open it there than by the rectum. The instrument commonly employed is the trocar, and in using it care must be taken in the selection of the site for puncture, lest some of the adjacent viscera be injured. This care is the more necessary, inasmuch as the trocar must be pushed deeply into the sac, so as to penetrate the laminated coagula forming the outer boundaries, which are often of considerable thickness; all possible care also must be taken not to wound any large vessel and to prevent the access of air into the cavity, as this has proved to be one of the dangers besetting the operation, and in no case must the risks be increased by injections to wash out the cavity. Sir James Simpson recommended that the opening should be made with a tenotomy knife and gradually enlarged with the fingers, so that the masses of clot might be discharged by the larger opening. When the opening is made cautiously in this way, there is probably little risk of serious hæmorrhage from the edges of the wound; but the objections are, that there must be a large access of air into the cyst cavity, and it is doubtful whether artificial opening is called for unless the contents have become so liquid that they will run through a trocar. In some cases no doubt the aspirator would prove a very useful instrument. In any special instance where it seems desirable to enlarge the opening, this can always be done after the use of the trocar or aspirator.

The management of patients suffering from hæmatocele in its chronic stages, when no surgical treatment is adopted, equally deserves the careful attention of the medical practitioner. The therapeutic indications are to prevent as far as possible the recurrence of active symptoms, and to promote the absorption of the extravasated blood, with the adventitious products which have followed in its train. For some months, therefore, after the acute symptoms have passed, the patient should have absolute rest at the catamenial periods, and in the inter-menstrual intervals the amount of exertion should be carefully regulated: no great efforts or straining should be permitted, and all excitement of the sexual organs should be avoided. It is necessary to see that the bowels act easily and regularly, and the diet should be nutritious, but not over-stimulating. If there is anæmia, and this associated with continued dribbling discharge of blood from the uterus, acid chalybeates and other tonics may be administered. To aid in dissipating tumour or thickening, the syrups of iodide and bromide of iron may be given; and locally, the application of blisters, of tincture of iodine, mercurial and belladonna ointments may prove serviceable.

The same precautions should be observed when artificial evacuation has been practised or spontaneous discharge has taken place. In these cases besides, great care must be taken to ward off the threatenings of fresh peritonitis, and to support the patient's strength if there is evidence of failing power from profuse discharge, or of grave depression from septicæmia. If there be signs of fresh suppuration after the use of the trocar, with renewed distension of the cyst, the laying open by larger incision will be almost inevitable, and this will then give most relief to the patient.

In all cases continued care and watching will be required long after the necessity for all active treatment has ceased, and the avoidance of all great exertion should be recommended, while change of air and proper hygiene should be prescribed by way of perfecting the recovery of the patient.

BIBLIOGRAPHY.

Récamier, Lancette Francaise, July 1831.
Bourdon, Revue Médicale, 1841.
Velpeau, Recherches sur les Cavities Closes, 1843.
Bernutz, Archives de Medicine, 1848; and Diseases of Women, by Bernutz and Goupil, Syd. Soc. Trans.
Piogey, Bull. de la Soc. Anat., 1850.
Vigues, Des Tumeurs Sanguines de l'Excav. Pelv., 1850.
Nélaton, Gazette des Hôpit., 1851.
Fénerly, Thèse inaugarale, 1855.
Robert, Bull. de la Soc. de Chir., 1851; and Gazette des Hôpit., 1855.
Huguier, Lecture before Surg. Soc. Paris, 1851.
Follin, Gazette des Hôpit., 1855.

Laborderie, Gazette des Hôpit., 1854.
Gaillard, Union Médicale, 1855.
Monod, Bull. de la Soc. de Chir., 1851.
Voisin, De l'Hématocele Rétro-Uterine, 1858.
Laugier, Comptes Rendus, vol. xl.
Engelhard, Archiv. de Médic., 1857.
Rouget, Journ. de la Physiol. de l'homme, &c., 1858.
Puech, De l'Hématocele peri-uterine et de ses sources (Montpellier, 1858).
Scanzoni, Diseases of Women, Amer. Edit.
Braun, Weiner Med. Wochenschr., 1861.
Ferber, Arch. f. Heilk., 1862, No. 5.
Crédé, Monatschrift, f. Geburtskund, vol. ix.
Breslau, Monat. f. Geburt. &c., vol. ix.
Seyfert, see Tuckwell's Work.
Dolbeau, Med. Times and Gazette, 1873.
Oldhausen, Archiv. f. Gynak, 1870.
Tilt, Diseases of Women.
Matthews Duncan, Edin. Med. Journ., 1862 and 1865.
Barnes, St. Thomas's Hosp. Reports, 1870; and Obstet. Trans. &c., vol. xiii.
Tuckwell, On Effusions of Blood in the neighbourhood of the Uterus, 1863.
Meadows, Obstet. Trans., vol. xiii.
Madge, Obstet. Trans., vol. iii.
West, Diseases of Women.
Simpson, Sir J. Y. Diseases of Women.
M'Clintock, Diseases of Women, 1865, &c.
F. Weber, Berlin Klin. Wochenschr. No. 1, 1873.

PELVIC CELLULITIS—PELVIC PERITONITIS.[1]

BY WILLIAM OVEREND PRIESTLEY, M.D., F.R.C.P.

DEFINITION.—Inflammation of the cellular or connective tissue, and of the peritoneum subtending the uterus and its appendages in the female pelvis.

NOMENCLATURE AND HISTORY.—Much confusion exists concerning the nomenclature of these affections, and a diversity of appellations has been employed by authors to designate them, in accordance with the views entertained of their pathological nature. Thus the terms "peri-uterine cellulitis" (Thomas), "para-metritis" (Virchow), "peri-uterine phlegmon" (Nonat), have been applied to designate inflammation of the connective tissue in the pelvis; "pelvic peritonitis" (Bernutz), "peri-uterine inflammation" (Courty), "peri-metritis" (Virchow), to indicate inflammation of the peritoneum covering the organs in the pelvis. It is only lately that inflammations occurring in the pelvis outside the uterus and its appendages have been recognised as distinct affections; and still more recently that differences have been pointed out between inflammations in the connective tissue and inflammations of the peritoneal coverings.

The formation and bursting of abscess in the pelvis seems to have attracted the attention of the oldest authors. Among others of Hippocrates, Galen, Oribasius, and Paulus Ægineta. These authorities however did not distinguish between abscess of the uterus and surrounding structures. During the last century "pelvic abscess" attracted the attention of several surgeons and obstetricians, and it was by them distinguished from abscess occurring in the parietes of the viscera contained in the pelvic cavity. Delamotte, Ledrun, Bourienne, &c., wrote on this subject as a surgical affection; and Levret, Puzos, and Delcurye directed attention to abscess forming in the female pelvis subsequent to delivery, under the title of "Depôts laiteux;" these authors being impressed with the notion that the formation of pus in this region was the result of a metastasis of milk, the breasts of those women who were the subjects of this complaint being observed either to secrete no milk at all, or to become flaccid previous to the appearance of suppuration. La Motte again considered puerperal pelvic abscess as produced by suppression of the lochia, and relates some interesting examples to illustrate his view.

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[1] This article was written in 1872.

Cases of abscess in connexion with the puerperal state which evidently belong to this category may be found scattered through the medical literature of our own country, corresponding to about the same period. Thus Smellie, William Hunter, and Dr. Denman have severally recorded cases of abscess following delivery, but these authors believe them of rare occurrence.

In the early part of the present century somewhat frequent contributions were made to the pathology and treatment of pelvic inflammations in women, but the writings on this subject were chiefly confined to the one phase—that in which abscess was already formed—and other pathological results escaped notice. In 1827 Dance and Husson published *Memoires sur quelques Engorgements Inflammatoires qui se developpent dans la Fossa Iliaque.* Melier, Téallier, Libeitard, Piotay, Fichot, Grisolle, Bourdon, severally followed in the wake of these. About the same time Dupuytren and Velpeau noticed this affection in lectures, and Martin of Montpellier, and Marchal de Calvi, wrote elaborate and valuable memoirs on the subject.

In Great Britain, a series of cases of pelvic abscess was published by Mr. Wainwright in 1841, and in 1844 a second series by Dr. Lever of Guy's Hospital. Besides these, detached instances were put on record by Dr. Collins, Charlton, and others. The first comprehensive attempt in this country to elucidate the pathology of pelvic inflammation and to point out its comparative frequency, not only after delivery, but in non-puerperal patients, was by Dr Doherty in a paper on "Chronic Inflammation of the Appendages of the Uterus;" and this was immediately succeeded by an excellent essay "On Abscess of the Uterine Appendages," by Dr. Churchill.

About the same time Sir J. Y. Simpson made "pelvic cellulitis" the subject of lectures in the University of Edinburgh, and seems to have originated the appellation. Soon after this, chapters on pelvic inflammation, under the titles of "Cellulitis," and of "Inflammation of the Uterine Appendages," found their way into the systematic works of Henry Bennet, Churchill, and West, in this country, and of Gaillard Thomas in America.

The most recent contributions on the subject are by Drs. Charles Bell and McClintock; MM. Gaillard, Nonat, Bernutz, Aran, and Courty; Virchow, Buhl, and Klob. Dr. Matthews Duncan has lately published an excellent and exhaustive monograph, which discusses all the latest pathological views. He proposes that we should discard former synonyms as imperfectly defining the nature of the two varieties of inflammation in the vicinity of the internal generative organs in women, and employ only the terms suggested by Virchow "perimetritis" and "para-metritis," the former to indicate inflammation of the peritoneum covering the uterus and its appendages, and the latter the inflammation of the cellular tissue. These appellations are however not free from all objection, and it has been thought best in this essay to adhere to the terms "pelvic cellulitis" and "pelvic peritonitis." These have taken root and are understood by the profession, while confusion is likely to increase from a multiplicity of names, and it is futile to expect to find terms which without change will express the varying views taken of the pathology of these affections. An additional reason exists for abiding by the terms "pelvic cellulitis" and "pelvic peritonitis"—in the fact, that these are adopted in the new "Nomenclature of Diseases" drawn up by the Royal College of Physicians of England, and approved by the Registrar-General.

It is convenient to study both affections together, as they have in a great measure the same causes, approximatively the same symptoms, and require to some extent the same treatment, while they are often associated together;—one by contiguity lights up the other, and they may pursue their respective course at the same time and in the same patient.

It is not proposed in this essay to include the forms of pelvic inflammation or abscess, other than those occurring in relation to the female generative organs.

PATHOLOGY AND MORBID ANATOMY.

I. PELVIC CELLULITIS.

When inflammation attacks the cellular tissue in the neighbourhood of the uterus, it is generally by extension from some portion of the generative apparatus. The cellular tissue in parts of the pelvis surrounding the uterus and its appendages, is very considerable in quantity, and when inflammatory products are formed, exudation takes place into its meshes, in proportion to the intensity and extent of the inflammatory action, and the laxity of the structures involved. The connective tissue is found uniting the several pelvic organs beneath the peritoneum, and exists in abundance between the vagina and rectum, between the uterus and the bladder, and between the folds of the peritoneum which constitute the broad ligaments of the uterus. From these localities it passes by continuity between the rectum and sacrum behind, and upwards it passes into the iliac fossa and along the surface of the psoas muscles posteriorly, and between the peritoneum and transversalis fascia in the anterior part of the abdomen. The peritoneum is so closely adherent to the anterior and posterior aspects of the uterus, that the cellular tissue there is sparing in quantity. The rectum, vagina, and urethra are each enclosed in a tube of pelvic fascia, and in normal conditions are enabled to glide about in their respective sheaths in the performance of their functions, through the loose attachment of the connective tissue about them.

Some French authorities have endeavoured to draw a distinction between inflammation of the cellular tissue immediately surrounding the cervix uteri, and that enclosed in the folds of the broad ligaments which stretch out laterally to the walls of the pelvis, and have described them as separate pathological affections. This separation, scarcely called for by the circumstances, is more likely to lead to confusion than clearer definition of the subject, and practically is of no great value.

It is obvious that the system of connective tissue may become inflamed by extension of mischief from the intestine, pelvic or vertebral bones, or other structures apart from the generative organs. These varieties of cellulitis however are not gynæcological affections and are not included in this article.

It is important to remark that pelvic cellulitis occurs both in puerperal and non-puerperal women, and is not necessarily synonymous with pelvic abscess. Suppuration is only *one* of the results of cellulitis, and it has been pertinently remarked that it would be as incorrect to apply the term empyema to all cases of pleurisy as to give the appellation of "pelvic abscess" to all cases of "pelvic cellulitis" because suppuration is one of the occasional terminations.

The pathological results are—1. Congestion and the effusion of serum; 2. The exudation of coagulable lymph; 3. The formation of pus.

1. The effusion of serum is the first and earliest product of the inflammatory action after it passes beyond the stage of mere hyperæmia. The infiltration of fluid exudation through the meshes of the connective tissue gives rise to swelling of a more or less elastic character in some portion of the pelvic cavity, which may be detected if examination is made sufficiently early. Even at a more advanced period, when the centre of the swelling is hard and firm from denser exudation, an outer and somewhat softer circle may be felt of serous effusion. Examples of this kind of effusion also sometimes present themselves in the œdematous swelling of the recto-vaginal septum when the circulation above is obstructed by the presence of either hæmatocele or inflammatory phlegmon. In some patients of feeble power and impoverished blood, the inflammatory action has no tendency to go beyond the stage of serous effusion, which may nevertheless be of considerable quantity and form a bulky tumour in the pelvis. Sir James Y. Simpson relates an instance in which a considerable quantity of serous fluid was drawn off with an exploring needle, which coagulated as it grew cold. Unfortunately there is no sufficient proof from the history of this case that the fluid was not an encysted serous collection of fluid in the peritoneum, and as we shall see presently, the more recent investigations of M. Bernutz and Dr. Matthews Duncan throw doubt on this point. There is abundant proof nevertheless that serous effusion does take place in the connective tissue about the uterus and its appendages. The semi-elastic swelling thus produced may be observed in some

instances gradually extending from an irritable ovary or from the uterus in a state of chronic inflammation, whenever hyperæmia exceeds certain limits and affects the surrounding parts, and it creeps along, sometimes rapidly, sometimes more slowly, distending the meshes of the cellular tissue until possibly it invades a large space in the posterior and lateral parts of the pelvis, and even runs up above the pelvic brim into the iliac fossa. When the loculi of cellular tissue, bounded by firm and unyielding layers of fascia, and denser subperitoneal connective tissue, become tense from effusion, the swelling acquires a sense of firmness so great that it may readily be taken for lymph deposit or for solid growth. The rapidity however of its subsidence when action abates, and the entire disappearance of thickening within a comparatively short space of time, as recovery progresses, forbid the inference that the tumefaction was due to exuded lymph, either in the connective tissue or on the surface of the peritoneum. The alternate development and recession of this kind of intumescence is very remarkable in some cases, and instances have been observed where the ovaries were so irritable that the approach of each catamenial period was attended by this kind of serous infiltration into the surrounding parts, and where it was absorbed habitually after the termination of the period.

Dr. West compares some of these forms of pelvic effusion to those inflammations of the cellular tissue which succeed to operations, and which have been named, by the distinguished Russian surgeon Pirogoff, "acute purulent œdema." He describes the fluid as a thin sero-purulent matter, which often retains its character long after it has formed in quantity sufficient to impart to the fingers a most marked sense of fluctuation. This analogy may hold good in a certain proportion of cases, but there are doubtless besides forms of œdema which are not purulent, as may be inferred by the rapid absorption and disappearance of the tumour.

2. The exudation of the coagulable lymph takes place whenever inflammation reaches the second stage, and this occurs in the majority of cases of cellulitis.

The amount of deposit so thrown out varies much in accordance with the acuteness of the inflammatory action and also with its duration. It may be so inconsiderable as to produce only some thickening in the pelvis, and render it difficult to determine whether the exudation is in the cellular tissue or on the free surface of the peritoneum, or it may be to such an amount that it completely surrounds all the pelvic organs, fills the pelvis, and mounts up as before stated by the continuity of the connective tissue into the abdomen or iliac fossa. When the tumour is very large and projects above the pelvic brim, it will in most cases be found that the uterus itself is much increased in size and forms part of the mass, and also, as pointed out by MM. Bernutz and Goupil, and by M. Aran, that peritoneal adhesions are formed which unite folds and coils of intestines together and thus add to the bulk.

The most notable characters of the tumour thus formed are its hardness, irregularity, and immobility. The exudation being thrown out between the laminæ of fascia which traverse the pelvis in different directions, and extending commonly from the lateral margins of the uterus to the bony wall of the pelvis, completely fixes the womb, and the swelling feels as though plastic plaster of Paris had been poured in and hardened there. So firm is the lymphy deposit in some cases, that if at all rounded in form it may be taken for an exostosis in the pelvis. Dr. Doherty compared the thickness and density to that of a "deal board." The immobility and irregular form serve to distinguish it from fibroid, and ovarian tumours. At first the swelling may seem to be rounded in form, but as it extends it becomes more irregular in outline, and if its progress be watched from day to day, it may be noticed passing from the centre to the circumference of the pelvis, and, extending it may be in front or behind the uterus, it reaches over to the other side of the pelvis. The position of the swelling varies, and its shape and progress are modified by the arrangements of the fascia in the pelvis, and by the firm attachments of the peritoneum to certain portions of the pelvic viscera. The anatomical arrangements of the female pelvic fascia, and the way in which they influence the course of cellular inflammation here,—more particularly the direction taken by pus when formed,—have been carefully studied by M. Jarjavy in France, and by Drs. Priestley and Savage in this country. König has published a still later contribution on this subject, and he details experiments made by injecting air and water into the cellular tissue under the broad ligaments. He infers that inflammatory effusion must necessarily extend itself in definite directions pointed out. Interesting as these investigations are, it must be observed that practically they do not afford a very accurate guide as to the direction in which exudation may extend. In the travelling of fluids, such as serous effusion and purulent matter, the planes of fascia and close attachments of the peritoneum, exert a most important influence, and direct pointing to the least resisting part; but when inflammatory exudation is in active progress it readily passes by contiguity from one loculament of cellular tissue to another, until all is one fused mass, the layers of fascia opposing no efficient barrier. When a pelvic phlegmon localizes itself within moderate limits, so that it forms a definite tumour, its most frequent sites are between the cervix uteri and rectum, and in the direction of one or other broad ligament on the lateral aspects of the uterus. From these localities it may mount up and invade the loose cellular tissue in the iliac fossa, a continuous tumour extending thus from the true pelvis into the false. Occasionally the swelling is found between the uterus and the bladder; and in rarer instances it lies in front of the bladder altogether. When situated so far forward as this, it has generally extended from the centre of the pelvis, and having reached this locality may run upward along the cellular tissue, which exists abundantly between the transversalis fascia and peritoneum, until it reaches the umbilicus and forms a tumour in the anterior part of the abdomen.

3. *Formation of Pus.*—Suppuration does not commonly take place until ten or fourteen days after the onset of the attack, even in acute cases, and in the chronic forms the indications of its having occurred are often much later than this. The large amount of surrounding exudation often so entirely masks the evidence which fluctuation commonly affords of the presence of pus, that it may not be detected until long after it is formed. Frequently indeed it escapes by the rectum mixed with the stools before fluctuation has been discovered, partly probably because the pointing has been beyond the reach of the practitioner, and partly from the presence of surrounding lymph exudation. When suppuration occurs it does not necessarily take a diffuse form, but it begins, it may be, in a loculament formed by layers of pelvic fascia, which, if of sufficient density, may circumscribe the pus, and direct it towards the surface or canal by which it can be discharged. Occasionally suppuration is more general and a large pelvic abscess is formed, the dissepiments being broken down, and the loculi communicating one with another. Cases have been observed where suppuration, beginning in the true pelvis, has mounted up into the iliac fossa; or where it has begun in the iliac fossa, and passed down by the cellular tissue of the meso-rectum into the pelvic cavity. Suppuration has been seen to extend from the pelvis so as to occupy the whole iliac fossa on one side and reach upwards as high as the kidney. Many examples have been recorded, where suppuration has extended from the front of the pelvis upwards behind the abdominal walls—evacuation taking place near the umbilicus. Again, two or three centres of suppuration may be formed, each bound by its respective laminæ of fascia or dense peritoneum, and these may point in different localities, each abscess having a separate opening and bursting at different times.

Mr. Spencer Wells saw a lady in whom a pelvic abscess discharged not only through the rectum, the bladder, the vagina, and the loin, but gravitated down the leg, to open in the calf.

In some rare cases suppuration has been so extensive and destructive as to lead to sloughing of large portions of the pelvic areolar tissue, and Dr. M. Duncan, in the work previously alluded to, devotes a chapter to this subject.

Pointing and evacuation of pus may take place by the external surface of the body, by the bowel, by the vagina, by the bladder, or urethra, and in rare cases by the uterus and Fallopian tubes. In still rarer instances, the outlet has been through the floor of the pelvis near the anus, or through the obturator or sacro-ischiatic foramina.

In abscess following delivery evacuation probably takes place most frequently through the abdominal walls. In thirty-seven puerperal cases observed by Dr. McClintock, twenty abscesses discharged through the skin in the iliac regions; two above the pubes; one in the inguinal region; and one beside the anus; of the remaining thirteen, six were discharged per vaginam, five per anum, and two burst by the bladder. In the non-puerperal variety Dr. McClintock believes it is extremely rare for an abscess to discharge externally. In the non-puerperal variety evacuation most frequently takes place by some portion of the intestinal canal. The position of the tumour is more frequently observed in the posterior half of the pelvis than in any other locality, and if suppuration takes place the whole outer surface of the rectum can be surrounded by the pus, on account of the loose character of the cellular tissue about it, and as the bowel presents only a thin wall between the abscess and mucous tract, it is readily perforated. Occasionally the perforation is higher up, in the sigmoid flexure of the colon. The next most frequent channel for evacuation internally is by the vagina, and after this, by the bladder. Instances are rare where an opening has been effected through the walls of the uterus or Fallopian tubes. The thickness of the uterine walls, their resisting character, and the way they are still further enforced everywhere by the close adhesion of the dense peritoneum on the outer surface, except on the lateral margins, accounts for this rarity; but examples have been recorded by Lisfranc, Hawkins, Battersby, Marchal de Calvi, and others. Bursting into the peritoneal cavity is also fortunately as rare as it is most disastrous. The peritoneum lining the pelvic cavity is much thicker than that covering the intestines or other abdominal viscera. It is also so enforced by strong fibrous tissue in this region, that it may well act the part of a fascia, in offering resistance to, and directing the contents of an abscess beneath. In most cases of inflammation too it is still further enforced by plastic exudation on its free surface, and thus its integrity is preserved.[1]

The following notes and results of the autopsy in the case of a young woman who died from the effects of a pelvic abscess following abortion, illustrate the pathology of the subject. The immediate cause of death was the exhaustive diarrhœa produced by the constant discharge of purulent matter into the intestine.

Before death an indistinct swelling, about the size of an orange, could be felt through the abdominal walls at the back of the pelvis. On examination, per vaginam, a tumour of irregular form was found behind the uterus. It extended from side to side in the upper part of the concavity of the sacrum, and was most prominent in the median line, where it bulged forward the cervix uteri and upper part of the vagina. The uterus was pushed altogether somewhat towards the symphysis pubis, and the rectum was compressed into a flattened form behind, being everywhere surrounded by hardened deposit. No fistulous opening could be reached by the examination per anum. The whole structures at the back of the pelvis were fixed and immobile, and the uterus was as fixed behind as if encircled by malignant infiltration. The ease with which the finger could be passed through the rectum excited attention. Its calibre was curiously preserved, considering the denseness of deposit around it. The body was much emaciated and very anæmic. On laying open the abdominal walls, there was no sign of inflammation until the intestines were removed from the pelvis. Then the coils lying lowest and posteriorly were found to be injected and slightly adherent to each other, and to the peritoneum beneath. A small quantity of serum containing flakes of lymph was also observed in this locality. The plastic exudation covering the intestines had all the indications of being recent, and its adhesive power was so low that the adhesions readily broke down. The entire uterus was pushed forward by a swelling on its posterior aspect, the bulk of which was about that of a medium sized orange. This was distinctly beneath and outside the peritoneum, and there was no difficulty in tracing the continuity of that membrane over the surface of the tumour, although it was covered with flakes and patches of lymph, and here and there crossbands of soft adhesion passed from it to the intestines and sacral peritoneum. On cutting into the tumour it was found to be partially but not entirely filled with offensive pus, intermingled with fæcal matter. An aperture, large enough to admit a crow's quill, communicated high up with the middle third of the rectum, and was at the upper part of the abscess, so that no proper drain of its contents could take place, but some of the contents of the bowel had passed into it. The cavity was regular in form, large enough to hold from one to two ounces of pus, and was lined with an ashy-looking pyogenic membrane. The uterus was enlarged and indurated, and its posterior wall almost as high as the fundus formed the anterior boundary of the abscess. The rectum and concavity of the sacrum formed the posterior boundary. Below, the mass was wedge-shaped, and almost reached the true pelvic fascia, a little below the level of the os uteri. Above, the peritoneum—its sub-cellular tissue much thickened by deposit—formed the roof. Everywhere around, but especially on the lateral margins, there was abundant hard plastic deposit, which fixed firmly the surrounding parts, and which creaked under the knife. The section looked like bands of dense fibrous structure with intervening pellets of fat. The ovaries and Fallopian tubes were slightly adherent to surrounding parts, but were otherwise healthy.

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[1] For illustrations of other rarer forms of opening and discharge of pus, see *Edin. Month. Med. Journ.* 1854.

II. Pelvic Peritonitis.

1. Plastic Exudation.—Some of the minor pathological results of pelvic peritonitis are sometimes remarked in association with the progress of certain diseases of the pelvic organs, as, for example, the dryness and sense of friction which may be detected with the hand and stethoscope over the surface of fibroid and ovarian tumours, when

the earlier stages of inflammation are in progress or have passed. Thickening and roughness on the peritoneal surface of pelvic tumours and over the surface of the pelvic organs generally, is not unfrequently observed in post-mortem examinations. The commonest effect of inflammation of the pelvic peritoneum is to throw out lymph exudation, which glues the organs together, and fixes them by adhesion. In this way the uterus becomes fixed to surrounding parts—most commonly by its posterior surface, where its peritoneal covering is most extensive, and then it is found to have lost its mobility in that direction, and to be inseparable from the rectum and sacrum. The tying down of the retroflexed uterus by peritoneal adhesions with such security that it is impossible to rectify the position either by finger or sound is familiar to most practitioners; and equally common is the fixing of the ovary from the occurrence of ovaritis, either to the structures near its natural position, or down in the recto-uterine cul-de-sac, into which it has previously sunk from its increased volume. The adhesions thus formed may so entangle and maim the various portions of the generative apparatus, as seriously to impede the performance of function. The ovaries and Fallopian tubes may be so surrounded by inflammatory deposit, that the elimination of ovules is obstructed, and menstruation is either suppressed, or is rendered painful and difficult. In like manner the fixing of the uterus may be a bar to impregnation, or if conception occurs, the expansion and ascent of the womb is prevented, and abortion takes place.

The plastic exudation of pelvic peritonitis sometimes glues together the pelvic organs and coils of intestine, so as to form a considerable tumour, which may, according to Bernutz, be mistaken for cellular phlegmon or some other pathological condition.

It may be remarked concerning the presence of these adhesions, that they may arise by extension of inflammation to the peritoneum from the cellular tissue beneath, and from the organs situated in the pelvis. They are produced also in the progress of cancer, and fatal terminations are thus in a measure averted. Further, in strumous patients there is a marked tendency to the association of tubercle with the inflammatory exudation, and thus pelvic peritonitis may become really a form of tubercular peritonitis.

2. Encysted collections of serous fluid in the peritoneal cavity have been described by various authors as pathological results of pelvic peritonitis. M. Huguier, in his paper "On Cysts of the Uterus, &c.," mentions two cases where "serous cysts of the uterus" were produced by metro-peritonitis, and these were recognised during life. The case recorded by Sir J. Y. Simpson, previously alluded to, in which a considerable quantity of transparent and coagulable fluid was drawn from a pelvic inflammatory tumour by an exploring needle, was probably of this nature—although Sir James believed the collection to be in the cellular tissue. Under the appellation of "encysted serous perimetritis" Dr. Matthews Duncan has described two instances occurring in his own experience in which he believes peritonitis led to the formation

of local collections of serous fluid, having all the characters of cysts, but the cyst-wall being formed "by the parietal peritoneum on the one side, and various parts of the bowels and uterus on the other side; these various parts being united by adhesions, as is frequently observed in cases of perimetric abscess and hæmatocele." The fluctuating tumours were at first supposed to be abscesses, but, when punctured, proved to have serous contents. As Dr. Duncan's patients recovered, no opportunity was afforded of verifying the diagnosis, and determining the actual position of the fluid, but the reasons advanced in favour of his view of the nature of the cysts are sound and precise, leave little doubt concerning the accuracy of his inferences. Dr. mas, of New York, also describes a case in which he drew off one or two ounces of serum produced by pelvic peritonitis. Limited collections of serous fluid in the peritoneal sac have been observed in connexion with the low form of peritonitis associated with malignant disease of the uterus and adjacent parts. An excellent example of s kind is recorded by Dr. Fiorget of Strasbourg, and quoted by Dr. Duncan. The author has seen on more than one occasion, limited collections of fluid formed in the lower part of the abdomen as the sult of tubercular peritonitis, which have a pathological relation to ose under discussion. The patients were young emaciated girls, the tumours so formed bore a close resemblance to cystic ovarian ease.

3. When suppuration takes place in the course of pelvic peritonitis, it may be at any part of the peritoneal surface where antecedent inflammatory products have been formed. Commonly the formation of pus is localized so as to form a limited abscess. If more extensive, the pus is contained in loculaments or cells which may communicate, or be separate, forming a series of distinct abscesses.

In position, the tumour so formed may be either between the uterus and rectum in the space of Douglas, or in some other part of the pelvic cavity. Baudelocque in his Traité de la Peritonite (1830), and Andral in his Clinique Médicale (T. ii., p. 722), have recorded cases of peritoneal abscess circumscribed to the recto-uterine cul-de-sac, and Bernutz and others have published similar cases. If above the true pelvis, it forms a tumour either in the hypogastrium or iliac fossa. Then adhesions and thickening from lymph exudation can commonly be traced down to the uterus and its appendages, indicating that the origin of the inflammation has been in the genital organs. Occasionally intra-peritoneal abscesses are formed at a considerable distance from the uterus, although the inflammation has originated there, and the track of the inflammatory progress is marked by lymphy exudation. Dr. Duncan watched a case where, during the progress of post-partum local peritonitis, a lump the size of an orange appeared between the navel and pubes, and which he believed to contain pus. The tumour was attached to the abdominal wall and moved with it, and between it and the pubes there was hardness, but not absolute dulness. The swelling soon disappeared, but no pus was ever observed in the stools, the inference being that the abscess burst into the upper portion of the alimentary canal. The doubt suggests itself, whether in this single case, the tumour, so quickly disappearing, was not a *serous* collection of fluid, speedily absorbed on bursting. Be this as it may, the occurrence of successive abscesses in different portions of a mass of lymphy deposit within the peritoneal cavity situated in the pelvis has been established, and each new centre of suppuration may give rise to a renewal of active symptoms. If the abscesses converge, they may unite and communicate freely with each other, the interior of the general abscess then representing a series of amygdaloid cavities,—not unlike the burrowings of a mammary abscess.

In the more extensive forms of suppuration connected with pelvic peritonitis, it is not uncommon to find one or both Fallopian tubes also distended with pus, and perhaps a separate purulent collection in the ovary, as well as in the cellular tissue of the broad ligament (Scanzoni).

The post-mortem appearances in pelvic peritonitis ending fatally, are well illustrated in the following typical case published by M. Bernutz, and in the subsequent description of parts on dissection by M. Aran:—

"On opening the abdomen, the bladder, uterus, broad ligaments and sigmoid flexure were all bound together by old firm adhesions. The posterior surface of the bladder was united to the uterus by two bands of adhesions—one of which passed on to the sigmoid flexure, the other united also the Fallopian tube to the sigmoid flexure. Between these two vesical bands, the vesico-uterine peritoneal cul-de-sac was healthy. The right broad ligament covered by the membrane from the bladder formed, as it passed behind the border and right angle of the uterus, a *demi-involucre,* which constituted the upper and internal wall of the pelvic cavity of that side. All this peritoneum was covered with false membrane. On the left there was no pelvic cavity, the broad ligament was united to both bladder and rectum; on quietly separating these adhesions, an intra-peritoneal abscess full of pus was opened; it was situated in front of, and below the ovary, being in direct contact with peritoneum covering that viscus. The uterus itself was bound posteriorly to the rectum, but was also acutely anteflexed. The Fallopian tubes were highly congested. The right contained two small purulent collections, one at the fimbriated extremity, which was dilated and firmly adherent to the ovary. The left tube was impermeable, but contained no pus. The ovaries were both healthy. The cellular tissue of the broad ligaments and uterus was perfectly healthy. If we set about," says M. Aran, "with care the dissection of the tumour, we find that it is constituted, proceeding from without inwards, by false membrane, still soft and pretty easily torn, forming a layer more or less thick, sometimes quite continuous, at other times hollowed out here and there by a certain number of locules full of a liquid, sometimes yellow and transparent, sometimes sero-purulent, or perhaps true pus, somewhat liquid and very serous. I have seen some of these cavities hollowed out in the false membranes, which contained a teaspoonful, sometimes even a tablespoonful, of purulent serosity or of pus. Underneath the false membrane we observe in the peritoneum of the subjacent parts, the evident marks of a recent inflammation, very fine and very close injection, thickening, and a serous infiltration of the subperitoneal cellular tissue, which allows the membrane to be easily peeled off. In the centre of the tumour we find perhaps one of the appendages of the uterus, perhaps the two appendages of one side,—ovary and tube—exhibiting some of the alterations which we have described *à propos* of acute inflammation of the ovary or of the tube, and the inflammation may extend as far as the broad ligament, the peritoneum of which may be very brightly injected, and the cellular tissue infiltrated with serosity, sometimes sanguinolent, more rarely purulent."

M. Bernutz, an author whose opinion deserves all consideration and respect, has laboured to prove, that the supposed inflammatory tumours of the cellular tissue in the pelvis, which have been described by various writers, are in reality misinterpreted cases of pelvic peritonitis, the swelling being produced by cohesion of convolutions of intestine to each other, to the uterus, and to the surrounding parts, and that when collections of matter are formed it is commonly inside the peritoneal cavity. He places himself in especial antagonism to M. Nonat, who has described inflammation of the pelvic cellular tissue under the appellation of "peri-uterine phlegmon," as of frequent occurrence, and expresses his belief, that while cellulitis is rare and never forms a bulky tumour, pelvic peritonitis is common, and has hitherto been mistaken for inflammation of the connective tissue. As in the case of peri-uterine hæmatocele, he compares the pelvic peritoneum in the female to the tunica vaginalis subtending the testicle in the male, and draws a parallel between the pathological results observed in the two serous membranes,—claiming an almost exclusively intra-peritoneal seat for the inflammation in both localities.

M. Aran, M. Courty, and Dr. Matthews Duncan so far agree with M. Bernutz, that they believe a *large* pelvic inflammatory tumour cannot be formed solely at the expense of the peri-uterine cellular tissue, and that when of any considerable size it is formed in the course of pelvic peritonitis by the adhesions of the various pelvic organs to each other. Both M. Aran and Dr. Duncan nevertheless attest the frequency of inflammation of the connective tissue, and Dr. Duncan seems to admit, that bulky tumours may be formed solely by thickening of the sub-peritoneal cellular tissue above the true pelvis.

To M. Bernutz is undoubtedly due much credit for the elucidation of the pathology of pelvic peritonitis. The subject had before his investigations been almost overlooked, or merged and confused with cellulitis. M. Bernutz has nevertheless pushed his conclusions too far, and in his contention has even misrepresented the opinions of others, notably of M. Nonat. In accepting a part of M. Bernutz's views, therefore, it is not necessary to impugn the accuracy of other observers, who attest the frequency of cellular inflammation and its results. The *relative* frequency of pelvic peritonitis, and of pelvic cellulitis, must be left for further investigation to determine.

CAUSES.—An important point in the etiology of pelvic-cellulitis and peritonitis has recently been discussed, viz., whether these affections are idiopathic, or have an independent origin, or whether they are always secondary, and result from extension of inflammation originally set up in some part of the genital apparatus. By Drs. Churchill, Bennet, Nonat, and others, they are described, in many cases at least, as separate or idiopathic affections, which may occur without preceding acute inflammation of the uterus or its appendages. M. Piotay first enunciated the view that inflammation of the uterus always precedes that of its annexes. M. Aran says that "the causes of peri-uterine inflammation are those of inflammation of the tube, and of the ovary, which is almost constantly the starting-point." Dr. West adopts the same view, and Dr. Matthews Duncan especially insists that peri- and para-metritis, as he terms them, are always secondary or the result of injury, and of all the prolific causes, inflammation of the mucous membrane of the womb, or endo-metritis, is the most common, both in the puerperal and non-puerperal states. The tendency of recent investigations is no doubt to show that the uterus, ovary, or Fallopian tube are the *points de départ* in the majority of cases, even if inflammation of the pelvic connective tissue and peritoneum are not exclusively propagated from these sources.

The causes therefore may be broadly stated to be those of inflammation of the uterus and its appendages: in the puerperal state, injuries produced by instruments during delivery, by turning, and by the pressure of the fœtal head on the maternal soft parts, when labour from any cause has been over protracted.

Irrespective of physical injuries, the peculiar "constitution of women" in the parturient state, illustrated by Dr. Charles Bell, is a strong predisposing cause to pelvic inflammation. Dr. West thinks that delivery and abortion stand in the etiology in the proportion of 77 to 100; and M. Bernutz states that about 44 per cent. are puerperal —the average between the two being about 60 puerperal cases in 100.

Special circumstances in the puerperal condition seem also to exercise an influence in the causation. Thus MM. Piotay and Grisolle affirm that primiparous patients are more predisposed than others to pelvic inflammation, probably because of the greater protraction and difficulty of first labours. M. Grisolle states that out of nine cases of pelvic abscess subsequent to delivery seven were primiparæ. The same author declares that women who do not suckle their children are more disposed than others to phlegmon of the pelvic cavity. He writes, that in every case he had observed following accouchement, "not one of the patients had suckled her own child, and the same appeared to be the case with every history transmitted by other authors." Further it has been observed that whenever the uterus contracts imperfectly after delivery, and is left abnormally large and relaxed in tissue, if the patient escapes general blood poisoning, there is a greater proclivity than usual to the development of metritis, and the extension of inflammation to the surrounding parts. Lastly, the getting up of the patient, the resuming of her occupations, or returning to her husband's bed, too soon after delivery or abortion, and before the generative apparatus has fully recovered the effects of the puerperal state, are fertile sources of pelvic inflammation.

Among the causes in the non-puerperal woman may be enumerated,—sudden suppression of menstruation from cold or other causes; mechanical injuries, either from accident or surgical interference; divisions of the cervix uteri; amputations; attempts to enucleate fibroid tumours; the use of intra-uterine pessaries; of laminaria and sponge tents; the application of escharotics to the cervix; and injections into the uterine cavity; the injudicious or rough use even of the ordinary instruments for diagnosis in uterine cases, may all be followed by pelvic inflammation. In the typical case of pelvic peritonitis recorded by M. Bernutz, the post-mortem results of which are given in a previous page—the inflammation was provoked by the mere passing of an uterine sound. The employment of strong vaginal injections may also stir up these forms of inflammation, and when the cervical canal is unusually patent, even injections of slight potency may run along the Fallopian tubes and produce serious symptoms. Further it must be added that in sensitive women coitus in early marriage, or its too frequent repetition at any time, may set up inflammatory action; and this is more readily developed when there has been some pre-existing uterine disease.

Authors agree in attributing to gonorrhœa an important share in the production of pelvic inflammation. Of M. Bernutz's 99 non-obstetric cases 28 were blenorrhagic. The study of these forms is interesting, inasmuch as they illustrate the way in which inflammation of the uterine mucous membrane in endometritis may extend itself along the Fallopian tubes, both to the ovaries and pelvic peritoneum. It may be stated that in this class of cases the form of affection assumed is much more frequently that of pelvic-peritonitis than cellulitis, and it is one of the most intractable and frequently-recurring forms.[1]

Dr. Duncan dwells on the importance of recognising the secondary character of pelvic cellulitis and peritonitis, more particularly as the pre-existence of endometritis may be overlooked on account of the more prominent symptoms of the secondary affection, and he moreover believes that swelling and tenderness of the ovary which often

[1] On this subject see a recent contribution by Noeggerath.

precede cellulitis or peritonitis, are due primarily to an extension of inflammation from the uterine cavity.

Lastly it may be mentioned that uterine fibroid tumours, uterine displacements, pelvic hæmatocele, malignant disease of the generative organs, and tubercular disease, may in their progress be attended by intercurrent cellulitis and peritonitis, and in cases of cancer, peritonitis is sometimes present, without the malignant disease having distinctly reached the surface of the peritoneum.

SYMPTOMS AND PROGRESS.—Cases of pelvic-cellulitis and peritonitis in regard to their symptomatology range themselves in two great classes. In the one class the attack is acute and the symptoms both general and local are well defined. The acute form is commonly ushered in with a rigor, and this is followed by fever, with increased temperature, flushing of the countenance, furred tongue, and an accelerated pulse. The urine is scanty and loaded with urates, and there is often pain and difficulty in micturition. Sharp pain is complained of in one or other iliac region or in the hypogastrium, and firm pressure affords no relief, but aggravates the uneasiness. A sense of dragging is often experienced about the umbilicus, and attempts to walk or even to sit upright, bring increase of distress. Following delivery or abortion, the first indications not unnaturally raise the suspicion of coming puerperal fever with general peritonitis; and it is only by observing the localization of the inflammatory action as the attack progresses, that its true character is ascertained. As in the more undoubted forms of puerperal fever, the breasts may become suddenly flaccid, or the secretion of milk from the first be arrested, and the lochia suppressed. In some of the forms at least, more particularly those which do not result from mechanical injury, there is an alteration in the blood analogous to that existing in puerperal fever and phlegmasia dolens, but the morbific action is less in degree than in the more serious forms of puerperal fever, and the results are more distinctly localized. In an acute attack of pelvic-peritonitis it is nevertheless not uncommon to find the abdomen tympanitic as well as exquisitely tender; the thighs flexed on the belly and the countenance anxious as in cases of general peritonitis. In the graver cases there may be severe headache and other forms of cerebral disturbance amounting even to delirium, and sympathetic vomiting may greatly add to the distress of the patient. Then, as in general peritonitis, the temperature instead of being above may be below the normal standard, indicating a condition, as pointed out by Wunderlich, always suspicious and full of peril.

In the slow or chronic forms, the advent of the disease is accompanied by symptoms much less prominent, and sometimes it may be so insidious as almost to escape observation, or at least to be misinterpreted. As before stated, both adhesions of the pelvic organs, and masses of inflammatory exudation in the cellular tissue, are frequently found on vaginal examination, the exact date of which it

is difficult to determine, because their formation has been attended by no sufficiently grave or definite signs. As early as 1843 Dr. Doherty called attention to the slow and insidious progress of chronic inflammation of the appendages of the uterus, which might end in abscess without any very prominent symptoms, and which sometimes occurred after the period when the puerperal woman is usually considered most obnoxious to inflammatory attacks. In the chronic form nevertheless, both in the puerperal and non-puerperal woman, although there may be no distinct rigor or pyrexia, the patient ordinarily complains of deep-seated uneasiness in the pelvis, combined it may be with increasing dysuria and constipation, and there is gradual failure of general health.

Such is the obscurity of the symptoms accompanying chronic pelvic-peritonitis after abortion or delivery that they are apt to be misinterpreted. Often the one prominent feature in these cases is grave and abiding discomfort in the region of the bladder, with frequent and painful micturition, and as the urine frequently contains pus, the symptoms are liable to be attributed to chronic inflammation of the bladder. Nevertheless a more careful investigation will prove that greater constitutional disturbance is present than the mere affection of the bladder will account for. There is anxious countenance, general wasting, inability to take food, and febrile disturbance which is indicated by a rise both in the pulse and temperature. Moreover tenderness on pressure over the hypogastrium extends beyond the limits of the bladder. Vaginal examination further discloses the fact that a partial or complete roof is formed to the pelvis, by exudation and by adhesion of all the structures there, and it is then obvious that the bladder affection is only a secondary complication of a more general pelvic inflammation.

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Pains in distant parts of the body, and sympathetic forms of disturbance, pelvic cellulitis and peritonitis have, in common with closely allied uterine and ovarian affections: in the chronic forms, habitual back-ache, with infra-mammary pain, pains in the mammæ, derangements of digestion, and the several forms of neuralgia, are inconstant and varying signs. The recurrence of vomiting in chronic cases is often very troublesome, and sickness is certainly most frequently present whenever the ovaries or peritoneum are involved. When a patient has to a great extent recovered, repeated attacks of nausea and sickness may occur if one or other ovary is yet bound down by adhesions, and impeded in the periodic performance of its functions.

In the chronic cases, where there may be very little constitutional disturbance and scarcely any local pain, the thermometer often rises to 101° or 102°, and it is a curious fact, repeatedly verified by observation, that even where a patient is so far recovered as to resume her occupations, the temperature, if taken at night, is often maintained one degree above the normal measure so long as any inflammatory deposit remains unabsorbed.

If suppuration takes place there is commonly an exacerbation of symptoms. When the case has been acute from the first the more active symptoms may have perhaps subsided for a while; but now there is a reaccession of shooting pains, with pyrexia, and these probably preceded by a rigor. When the case is chronic and sluggish in type, the occurrence of suppuration may be indicated by the supervention of pseudo-acute symptoms. Eventually the establishment in both cases of recurring signs of hectic, with the facial expression characteristic of the formation of pus, afford strong presumptive evidence of its occurrence even when this cannot readily be verified by physical examination.

The chief physical characteristics of tumour formed in the progress of pelvic cellulitis and peritonitis are, as previously pointed out, *irregularity*, *hardness*, and *immobility*. The stage of partial softness or elasticity is commonly so short as rarely to be recognised. If sufficiently large the swelling is felt above the brim of the pelvis by external examination; but more frequently it is limited to the deeper parts of the pelvic cavity, and internal exploration is required for its detection. The amount of inflammatory deposit indeed varies greatly. Sometimes it is only just enough to produce slight swelling and hardness on one border of the uterus, or to glue the uterus posteriorly and interfere with its mobility. Again, hardness and immobility without much sense of tumour may be confined to the roof of the pelvis, and give the impression of a thin plastic layer having been poured over the surface of the pelvic organs which has hardened there, fixing the uterus to the surrounding parts. This is characteristic of a pure form of pelvic peritonitis. At other times the exudation is so considerable that it fills the posterior half of the pelvis and pushes the whole uterus forward against the symphisis pubis. When swelling is circumscribed to the cul-de-sac of Douglas, it pushes the cervix uteri forwards and upwards, as in retro-uterine hæmatocele, the fundus being more or less retroverted. This displacement may be to such an extent that it is difficult to reach the os uteri. If formed in the lateral aspects of the pelvis it displaces the uterus over to the opposite side, and probably raises it towards the iliac fossa. When the tumour is large enough to reach above the pelvic brim, it will in some instances be found that its upper part consists of the fundus uteri pushed forward and upward by the inflammatory exudation in the lower and posterior parts of the true pelvis; and again it should be remembered that in other cases where the tumour attains large dimensions in the abdomen, it is formed in part at least by matting together of the intestines as the result of peritoneal exudation.

Whatever the amount or position of the inflammatory effusion, its tendency is to fix and unite the pelvic organs, and render any attempt to change their position with the finger impracticable. Some few cases only have been observed in which tumours of this kind have been more or less moveable. These were of very limited size, and generally limited to Douglas's cul-de-sac. In acute cases the parts are so tender that only the gentlest vaginal examination is tolerable. The genital passage feels hot; its arteries pulsate unduly, and occasionally the vagina is narrowed and distorted by the surrounding swelling impinging on its walls. If an examination be made by the rectum the results found on vaginal exploration are confirmed. The surrounding structures are fixed and immoveable, and the rectum is probably compressed by the tumour which lies between it and the uterus.

Whenever possible, external and internal examination should be combined, as when careful exploration is made of other pelvic tumours. In this way the size of the swelling may be best determined, and a distinction may be drawn between mere hardness or fixation and genuine tumour.

When suppuration has occurred fluctuation may not at first be readily detected. In some patients all the constitutional signs of suppuration are present, and yet the most practised touch fails to find any indication of softening in the accessible portions of the inflammatory tumour. Occasionally a pelvic abscess bursts into the intestine too high up to be reached by the exploring finger. In most cases, however, when abscess has formed, a softened or doughy feeling becomes eventually perceptible, in some part of the parietes, on examination either by the vagina or rectum, or external surface of the abdomen. In cases of doubt Sir James Simpson proposed to push a fine exploring trocar in the inflammatory tumour for the detection of pus; but this should only be resorted to when the general symptoms are urgent and threatening, and not merely for the purpose of confirming a diagnosis.

When evacuation of the abscess takes place an amelioration of all the symptoms usually follows, and the improvement in the condition of the patient progresses, unless purulent matter is contained in several separate loculaments, and the first discharge proves to be only the bursting of one of a series of separate abscesses. In this case only partial relief is afforded, and the hectic symptoms are repeated until all the abscesses are emptied.

Different estimates have been made as to the frequency with which suppuration takes place in proportion to the entire sum of cases of pelvic inflammation. M. Grisolle states that out of 73 cases, he had collected, resolution took place only in nine. Sir J. Y. Simpson held that suppuration occurred only in about half the cases. The value of M. Grisolle's statistics in aiding to form a general estimate is impaired by the fact that his cases were chiefly those in which inflammation was confined to the iliac fossa, and where causes other than those implicating the genital organs were concerned. More recent statistics tend to prove that suppuration is certainly more frequent in cases following delivery and abortion, than under other circumstances. Dr. McClintock gives the frequency of suppuration in post-puerperal cases at 37 in 70, and Dr. West at 51 in 100.

In the non-puerperal state, termination by resolution is the rule, and M. Gaillard places the frequency of suppuration under these circumstances at only 4 in 53 cases. The frequency of inflammation and abscess as affecting relatively the two sides of the pelvis has been an object of interest to investigators, and the estimates of various authors show great disparity. Taking the sum of cases recorded by Grisolle, Bourden, and Marchal de Calvi, the frequency is nearly double on the left side. In Gaillard's cases the preponderance was on the right, while in Dr. M. Duncan's cases, the frequency on the two sides was equal.

Although the opening and discharge of a pelvic abscess, commonly brings relief to the more urgent symptoms and convalescence progresses steadily afterwards; yet in exceptional cases the cavity of the abscess, instead of granulating up and being obliterated, becomes lined with a distinct pyogenic membrane, and although, perhaps, much contracted, continues to secrete pus for weeks or months after the first exit, through fistulæ which open either externally, or into some pelvic viscus. These pelvic fistulæ, by persistence, cause much discomfort, and at times become a serious drain on the strength of the patient. Simpson has recorded an instance in which chronic fistulous openings existed in the vagina, groin, and thigh, and it was found by injection that all communicated with each other. Whether a case terminates by resolution or suppuration, if there has been any considerable amount of lymph deposit during the progress of the affection, there will usually be indications of thickening to be found in the pelvis long after the subsidence of the attack, and the adhesions contracted among the various pelvic organs may be detected months after the patient is to all appearance perfectly well.

DIAGNOSIS.—The diagnosis is not ordinarily surrounded by any great amount of difficulty. From puerperal fever and general peritonitis, these affections may be distinguished by observing, as the attack progresses, the distinct localization of the symptoms, and the less severe character of the constitutional disturbance. In the later stages it is almost impossible to discriminate between the results of pelvic inflammation and hæmatocele, the local manifestations in both at certain periods of progress being almost identical. A careful inquiry into the history will, nevertheless, do much to clear up the differential diagnosis. In hæmatocele the attack is commonly developed during, or about, the time of a catamenial period, which probably is unduly profuse, showing a tendency to hæmorrhage, or else it is suddenly suppressed; the tumour is formed suddenly, and, if large, is accompanied with indications of anæmia, on account of the mass of blood which has escaped from the general circulation. The tumour again is most soft and fluctuating from the first, and hardens as time goes on. Lastly, the signs of local inflammation and of general febrile disturbance supervene after the formation of the tumour. On the other hand pelvic cellulitis and peritonitis most frequently succeed delivery and abortion; the tumours they form come slowly in comparison, are hard from the first, and may or may not soften in their later stages; local pain and febrile excitement distinctly precede the development of tumour.

The hardness and immobility produced by pelvic inflammation has repeatedly been mistaken for malignant deposit about the uterus and its appendages. The latter may be distinguished by noting that the symptoms have, probably, not followed parturition or abortion; have not been preceded by, nor are accompanied by, febrile action; that they are evidently an extension from an uterus affected by cancerous ulceration—the cervix being sound in most cases of pelvic inflammation. If further evidence is needed, the subsequent progress of the affection and the development of general cachexia leave no further doubt of the nature of the case, and it is always wise in doubtful cases to suspend an opinion until time clears up the difficulty. Ovarian and fibroid tumours may be diagnosticated by their slow growth, definite form, the non-occurrence of febrile symptoms, and generally by the absence of fixation; retroversion or flexion of the uterus in the unimpregnated condition by the use of the sound—in the gravid state by the general and local signs of pregnancy.

Great difficulties beset the diagnosis when a small ovarian or fibroid tumour, or a retroflexed uterus, is bound down by inflammatory adhesions and deposit in the posterior cul-de-sac; and the difficulty is the greater if inflammation is then in progress. In the case of retroflexion, a sound will indicate the direction of the uterine cavity when it can be safely used—although it may be impossible to replace the womb. When a small ovarian tumour is the centre of inflammatory deposit in the posterior part of the pelvis, its outline will become more sharply defined as time progresses, and it may probably be remarked, as the indications of inflammation subside, that a certain elasticity becomes apparent to touch, which presumably is not due to the softening of suppuration in the absence of the usual preceding signs of suppuration.

Extra uterine fœtation is to be distinguished by observing the previous signs of pregnancy, and the general progress of the case. The rupture of a tubal fœtation approaches nearer in its general symptoms to pelvic hæmatocele than to cellulitis or peritonitis. Fæcal accumulations cause frequent mistakes, and may, unless care be taken to secure a free evacuation of the bowel before examination, be erroneously taken for masses of inflammatory thickening. The diagnosis between abscess forming in the pelvis as the result of caries in the vertebræ, of the bones of the pelvis, or originating in connexion with some part of the intestinal track, must be made out by the antecedent and general symptoms of these conditions detailed in their own place.

M. Bernutz and Dr. Thomas have attempted to discriminate between the signs, both general and local, of pelvic cellulitis and pelvic peritonitis. Dr. Thomas indeed has published an ingeniously drawn up table of differences. Practically however it will be found that only in some few typical and rare cases are the symptoms and physical signs so dissociated as to enable the practitioner to say this is pure peritonitis and that pure cellulitis. The signs of one affection may predominate over the other, and give a type to the case, but in most instances the same symptoms answer for both, and rarely does the one form of inflammation occur without being complicated with, and merging more or less into the other. The chief points of difference dwelt upon by these authors are first, that the tumour is generally confined to the true pelvis in the case of peritonitis, but can commonly be felt reaching from the broad ligament to the pelvic brim and iliac fossa in cellulitis. In the peritoneal tumour there are more or less distinct and hard prominences to be felt by the vagina, in the direction of the cul-de-sac; while the tumour is smoother and more lateral in position in cellulitis. Suppuration is rare in the peritonitic form, frequent in cellulitis. The uterus is always displaced to the opposite side, even when it is immoveable, in pelvic peritonitis; not necessarily displaced, and interference with its mobility less marked, in cellulitis. Retraction of the thigh is rare in peritonitis, common in cellulitis. Constitutional signs of peritonitis are present in the one case, not in the other.

There is no assured method of distinguishing the serous collections of fluid from those of pus when occurring in the course of these affections without the aid of the exploring needle, which it may not always be expedient to use. The exploring needle may nevertheless be advantageously employed previous to evacuation when this operation seems called for, and a doubt remains concerning the nature of the swelling.

The Prognosis is generally favourable, but the duration is uncertain and variable, and the complications and perils are numerous. A large number of cases terminate by resolution, and if in these instances the symptoms are slight and the results do not go beyond the effusion of serum or soft plastic exudation, the patient soon recovers and the swelling speedily disappears. When exudation is extensive and dense, and there is much fixing of the pelvic organs, convalescence will be more tardy and the results will pass more slowly. Thus a patient may continue in feeble health and with impaired powers of locomotion for a considerable time. In these cases swelling and hardness may be found in the pelvis on examination for an unlimited period, and the fixed condition of the uterus interferes with the performance of its functions. Menstruation is rarely altogether arrested, but pregnancy with a fixed uterus is not very likely to take place; and even if conception does occur, there is proclivity to abortion, because the expansion and rising of the uterus are interfered with during its development. The tendency, of plastic exudation and of adhesion in the pelvis, however firm, is no doubt to disappear in the progress of time, like the adhesions formed in the pleura and elsewhere, and repair effected in this way is sometimes very remarkable. In some examples, nevertheless, the fixed condition becomes permanent and irremediable, and hence it happens not unfrequently that operations about the uterus followed by pelvic cellulitis and peritonitis leave such results that the reproductive apparatus is permanently maimed. It may be stated as a general proposition in reference to puerperal cases, that the prognosis is graver both in pelvic peritonitis and in cellulitis than in the non-puerperal condition, and further, the danger is the greater in proportion as peritonitis is the more predominant, inasmuch as there is then great tendency of inflammation to spread to the general abdominal cavity, and parturient women ill bear inflammations of serous membranes. Again, the formation of abscess occurs more frequently in association with puerperal cellulitis than under other circumstances, and brings its own perils.

Fortunately most patients in whom pelvic inflammation runs on to abscess incur little risk of a fatal termination, unless suppuration is very extensive or prolonged or other complications are added. Evacuation takes place either by spontaneous or artificial opening, and recovery follows. The dangers which beset such cases are of exceptional occurrence and can often be averted by judicious treatment. The sources of danger are first, that occasionally pelvic abscess bursts into the cavity of the peritoneum, and this is almost inevitably followed by collapse and death. The danger of this catastrophe is always greater when the abscess is intra-peritoneal, because the cavity is necessarily formed in some part of its extent by false membranes of little resisting power. Dr. McClintock, without distinguishing between pelvic cellulitis and peritonitis, avers that the danger of bursting into the peritoneum is greater in the non-puerperal than in the puerperal forms of pelvic inflammation. He records three cases occurring in non-puerperal patients, and had never seen a similar result in abscess succeeding to parturition.

Secondly, in cases where suppuration has been established and evacuation has taken place, the safety of the patient is sometimes jeopardized by the extent of the suppuration and its persistence. The non-contraction of a pelvic abscess after bursting and the continued discharge of pus for a prolonged period, through fistulæ, which may be single or multiple, and perhaps so inaccessible as not to be amenable to treatment, may eventually wear out the strength of the patient or develop tubercular disease. This form of peril becomes more prominent where fistulæ communicate with the colon or rectum and keep up an exhausting diarrhœa or dysentery, over which remedies have little control. Moreover, when suppuration is so extensive it often destroys the ovaries, renders the Fallopian tubes impervious, and possibly permanently displaces the uterus, thus irreparably damaging the reproductive apparatus, and arresting or disturbing its functions.

Thirdly, the occurrence of suppuration may be the occasion of septicæmia, and the patient may develop all the symptoms of general blood-poisoning.

Fourthly, cellulitis sometimes—but pelvic peritonitis more frequently—has a tendency in certain patients to assume a chronic type, and to take the form of repeated attacks, which may be determined by the occurrence of each catamenial period. In women so predisposed there is always a likelihood of tubercular peritonitis being set up, and the impaired nutrition and emaciation favour tubercular growth in the lungs and other organs, and thus the patient eventually dies of phthisis. If reference be made to the article on pelvic hæmatocele it will be seen that pelvic peritonitis is often the precursor of hæmatocele. MM. Tardieu, Bernutz, and others have carefully studied the relations between these two affections, and Virchow and Herber suggest the possibility of capillaries being formed in false membranes over the pelvic peritoneum and being the source of bleeding in such cases.

Lastly, sources of suffering and danger now and then present themselves in a less definite form, and on this account are more embarrassing to the medical practitioner. Chronic induration and deposit in the pelvis—in or outside the peritoneum—may become the seat of severe neuralgic pain, which increases with the feebler condition of the patient, and comes on in worse paroxysms as night approaches. The demand for opiates in some form or other, and in augmenting doses, increases, until it becomes doubtful whether the physical suffering of the patient or her morbid craving for the narcotic is the more formidable evil. In the meantime digestion and nutrition become impaired, the temper irritable and the mind enfeebled, and the patient either becomes a chronic invalid or dies of some form of tuberculosis. Further, if the ovaries are involved in chronic exudation, nausea and sickness are often added to the discomforts and perils of the patient, particularly at the menstrual periods; and in cases where inflammation, acute or chronic, extends to the bladder, the additional suffering so entailed, with the exhausting muco-purulent discharge from its mucous membrane, cannot fail, if prolonged, to be sources of danger.

TREATMENT.—The treatment of pelvic-cellulitis and of pelvic-peritonitis is the same as that for inflammation of the pelvic and abdominal viscera generally. In the acute form general blood-letting is now rarely practised, but leeches in moderate numbers, according to the urgency of the symptoms and the strength of the patient, may be applied without hesitation. They may be applied over the seat of pain, if this is above the pelvic brim; but perhaps in all cases more relief will be experienced when they are applied to the perineum or round the anus, than elsewhere, because there the vessels anastomose most freely with those involved in the inflammatory action. It is rarely advisable to apply leeches to the cervix uteri in acute cases, because the pain and tenderness preclude the use of a speculum or leeching-tube. Three or four leeches will commonly be sufficient, and at most five or six. The leeches may be repeated if necessary at longer or shorter intervals, but in the abstraction of blood even by leeches it is needful to bear in mind that the patient when attacked is probably in a depressed condition of health, and at the beginning of what may prove to be a protracted illness; hence much depletion may be a serious disadvantage later, and retard recovery. Light linseed poultices moistened with liniment of belladonna or laudanum, and kept constantly applied over the hypogastrium, afford much relief to pain; or when the weight of a poultice is ill borne, spongiopiline may be used as a substitute. It is well to avoid all purgative medicines and to keep the bowels quiet during the continuance of acute symptoms, and opiates in some form will be required to fulfil this indication and at the same time to relieve pain. Although much has been said and written against the employment of mercury, there is probably no series of cases in which its judicious administration in the early stage brings such obvious advantages as in those now under consideration. In combination with opium it seems to act as a direct sedative to inflammatory action, to lessen the tendency to plastic exudation, and to promote absorption when progress abates. With these objects calomel may be given in one or two-grain doses every four or six hours combined with half a grain opium, and if more opium be required it may be given at night, separately, either by the mouth, by suppository, or enemata, or by hypodermic injection.

Dr. M. Duncan prefers grey powder or blue pill, combined with Dover's powder or solid opium, to calomel and opium, which he has found to cause severe purging; but this disadvantage may, in most cases, be avoided by not giving too large doses of calomel.

In the lower forms of inflammation, particularly those which take a diffuse form, and are characterized by want of power, it is better to avoid mercurials and depletion altogether, and to trust to opiates alone; and in no case where mercury may be employed, should it be pushed to salivation, although the course may be repeated in case of relapse. If for any reason it is inexpedient to give opium in any of its forms, hydrate of chloral with henbane, belladonna, Indian hemp, or iodoform may be substituted, but none of these hypnotics are so trustworthy as opium or morphia.

Alsolute rest in the recumbent posture should be enjoined, and the patient placed in the attitude which gives greatest ease. The extremities should be kept warm, and the diet should be unstimulating, consisting of milk, beef-tea, and simple farinaceous articles. If there be marked general feverishness, salines, such as the acetate of ammonia or citrate of potash, may be given with small doses of aconite in the intervals of the doses of the mercurial with opiate, and if sickness is present, the saline may be prescribed in an effervescing form. Demulcent drinks, barley-water and toast and water, potash and seltzer-water may be permitted to relieve the thirst of the patient, and when bladder symptoms are troublesome, the decoction of *triticum repens*, recommended by Sir H. Thompson, may be prescribed. When vomiting is especially distressing it may be necessary to suspend other remedies for a while, until the stomach grows less irritable. To favour this quiescence small doses of hydrocyanic acid, either in effervescence or with carbonate of bismuth, or small doses of opium, or belladonna may be given. Sinapisms and blisters to the epigastrium have been found useful in some obstinate cases, and hypodermic injection of morphia combined with atropine has succeeded when other means have failed. In all such instances food must be given in most sparing quantity often repeated. Stimulants, even sparkling wines, are often hurtful; while ice with milk is retained and is grateful. Where even the simplest things are rejected, there is no alternative but to give nourishment by enemata.

When the sharper symptoms have abated, Dr. Gaillard Thomas strongly recommends the application every ten or fourteen days of a cantharides blister, four or six inches square, over the hypogastrium, as the most rapid and efficient remedial agent for promoting the removal of the exuded lymph and preventing the occurrence of suppuration. Valuable as such means may be, caution must be exercised in their employment, as in some sensitive women there is no doubt that cantharides blisters over the lower part of the abdomen provoke distressing and persistent strangury. When well borne by the patient there can be no doubt that blisters secure a more rapid improvement in the chronic and tedious forms of these affections, and to keep up a continued effect a small part of the blistered surface may be kept permanently open by dressing it with the French tissue-plasters known as "Albespeyres." Another method of producing counter-irritation which is free from the inconvenience of cantharides blisters, and may be used for a long period, is the painting over the surface above the pubes, once in twenty-four hours, with tincture of iodine; or, if thought desirable, equal parts of iodine and mercurial ointment may be applied instead. At this stage of the affection the bowels should be carefully regulated, and an occasional purge may be given if necessary; the diet should be somewhat improved, and the calomel and opium discontinued. If required, opium may be given alone, and the doses be at distant intervals. Instead of the febrifuge remedies, suitable doses of bromide or iodide of potassium with bitter infusion may be substituted three times a day; and if there is no great tenderness of the vagina, a warm douche may be used night and morning,—the injected water being slightly medicated with tincture of iodine or iodide of potassium, as recommended by Dr. Thomas. At no stage of the affection should the patient be allowed to quit the horizontal posture until movement can be effected without pain, and even when apparently convalescent, to prevent relapse, continued care and repose must be observed for two or three months afterwards at the return of each catamenial period. It has repeatedly been noticed that women who have left the hospital to resume their occupations apparently convalescent, are apt to have a reaccession of their original symptoms, and to seek readmission within a very short period. When indications of suppuration have shown themselves, either in the acute or chronic form, all forms of depletion, and all remedies which may possibly impair the strength of the patient, should be discontinued and a more tonic method generally should be pursued. With the first local or general signs of the formation of pus in the acute form, fomentations and poultices should be more sedulously applied to the lower part of the abdomen, and in the chronic type, where they have not previously been thought necessary or have for a time been discontinued, they should at once be brought into requisition. To envelope the whole pelvis in a large linseed poultice often gives the most marked relief to the suffering associated with this stage, and poultices may be aided if necessary by the local application of sedatives, or by sedatives given internally. These, with perfect rest and a careful regulation of a simple nutritious diet, attention to the state of the bowels, and generally to the various points in the management of the sick room, which so much conduce to the well-doing of all chronic invalids, comprise the treatment until abscess has formed.

Concerning the propriety of opening pelvic abscess there is considerable difference of opinion. The proper rule for practice, according to some authorities, is to evacuate the pus as soon as there is distinct evidence of its formation, or at least so soon after its formation as it is accessible to surgical procedure. By early opening, it is urged, the pain and irritative fever are cut short and cure is expedited. At the same time the danger of spontaneous openings taking place into the peritoneum or other inconvenient locality will probably be averted. M. Becquerel, on the other hand, goes so far as to assert that the abscess should never be opened artificially, and M. Aran affirms there is no evidence that artificial evacuation at one point prevents openings in other situations.

In truth there is no universal rule for practice in this matter. As in purulent collections forming elsewhere, the expediency of making an opening must be determined by the urgency of the symptoms, by the progress and duration of the case, by the accessibility to puncture, and by the probabilities of early spontaneous evacuation taking place. It is obviously undesirable to operate before the abscess is mature, or when it is so deep-seated as to render puncture hazardous and uncertain in result. In most cases spontaneous evacuation will take place at the best possible time and in the locality, considering all circumstances, sufficiently favourable without the intervention of the surgeon, and general treatment and local palliatives will only be required. When, however, the local and constitutional symptons are such as to cause anxiety; when, for example, pain is severe and constant, or when it returns in periodic paroxysms which entail much suffering; when irritative fever, colliquative diarrhœa, mucous membrane irritation,—as evidenced by stomatitis or sympathetic vomiting,—become so pronounced as to threaten serious exhaustion, then it becomes an obvious duty to promote as speedy an evacuation as may be possible.

Artificial opening may with less hesitation be undertaken, if pus is felt near the surface, either externally or internally, and fluctuation is distinct. Where it is deeper seated, and local examination is unable with sufficient precision to confirm the evidence of pus formation offered by the general symptoms, the use of the exploring needle suggested by Sir James Simpson may first clear up the doubt, and be followed by a wider opening. Often when a pelvic tumour has felt hard and inelastic, when the most careful *tactus eruditus* has failed to detect fluctuation, notwithstanding the general history of pelvic abscess, pus has been found to be present on puncture, and the deceptive hardness has been due to the great distension, and the thickened indurated condition of the cyst wall. In doubtful conditions of this kind the exploring needle or a fine trocar becomes an important aid both to diagnosis and as a preliminary to operation.

In operating, a Poiteau's trocar or guarded bistoury may be employed, but the bistoury is to be preferred, as the aperture thus made is wider and less likely to close before the whole of the purulent matter is discharged, than when a trocar is used. The puncture must necessarily be made where fluctuation is most distinct, or where there is most probability of reaching pus, always supposing that no special dangers are incurred in the locality so indicated. A careful examination must be made before puncture to ascertain that no considerable artery pulsates in the line of the proposed incision. When pus has once obtained egress there is no necessity to break down partitions or to inject water into the cavity of the abscess, as some have recommended. In abscess formed as the result of pelvic peritonitis, these procedures would in fact incur the peril of breaking down adhesions, which are the fragile safeguards against direct communication with the general cavity of the peritoneum.

When a choice is afforded of locality for incision, the general integument on the surface of the body is to be preferred. If the abscess is opened internally, the vagina is to be preferred for reasons previously stated, and the genital canal should then be gently washed out by some weak disinfectant injection once in twelve hours; more especially should this be done if the discharge is offensive in character.

The incision must nevertheless be made wherever the abscess is most easily accessible, and where its contents can most readily drain away after the operation.

In chronic abscesses which go on accumulating pus and repeatedly discharging, it may be necessary to use a drainage tube to secure a continuous escape of fluid.

For the treatment of persistent fistulæ the reader may be referred to Sir J. Y. Simpson's works.

The treatment of complications and general after treatment of patients who have been the subjects of pelvic abscess is identical with that which is appropriate for patients who have suffered from suppuration in other localities, and the various forms of persistent induration left as the sequelæ of pelvic cellulitis and peritonitis require the same measures as those prescribed for the chronic forms of uterine hypertrophy which are described elsewhere.

BIBLIOGRAPHY

Delamotte, Chirurgiæ.
Levret, Art. des Accouchements.
Ledran, Observations de Chirurgie, 1731.
Bourienne, Journal de Médicine, 1775.
Puzos, Mémoires sur les Dépôts laiteux, 1759.
Deleury, Traité des Accouchements, 1777.
La Motte, Traité des Accouchements, 1729.
Smellie, Midwifery, Vol. III.
William Hunter, M. S. Lectures, 1775.
Denman, Midwifery.
Dance and Husson, Mémoire sur quelques Engorgements Inflammatoires qui se Developpent dans le Fossa Iliaque Droit, 1827.
Mélier, Journal Géneral de Médicine, 1827.
Téallier, Ibid. 1829.
Libeitard and Piotay, Thèses Phlegmons de Fosses Iliaques, 1837.
Fichot, Thesis, 1839.
Grisolle, Phlegmons des Fosses Iliaques, Archiv. Gen. de Méd., 1839.
Bourdon, Revue Médicale, 1842.
Dupuytren, Clinique Chirurgicale.
Velpeau, Traité de l'Art. des Accouchements, 1835.
Marchal de Calvi, Abcès Phlegmoneux Intra-pelviens.
Martin, Jun., of Montpellier, Des Dépôts des Annexes de la Matrice, &c.
Wainwright, Med. Chir. Rev. 1844.
Lever, Guy's Hospital Reports, 1844.
Collins, Practical Treatise on Midwifery.
Charlton, Abscess of Iliac Fossa, 1841.
Doherty, Chronic Inflammation of the Appendages of the Uterus, Dublin Med. Jour., 1842.
Churchill, Abscess of the Uterine Appendages, and Diseases of Women, Dublin Med. Jour., 1843.
Simpson, Sir J. Y., Memoirs.
Bennet, Inflammation of the Uterus, &c.
West, Diseases of Women.
Battersby, Dub. Quart. Jour., 1847.
Bell, Charles, Edin. Med. Jour., 1856—7.

McClintock, Diseases of Women.
Gallard, Thesis, De l'Inflammation du Tissue Cellulaire, &c., 1855.
Nonat, Maladies de l'Uterus, and Gazette des Hôp., 1850.
Bernutz and Goupil, Diseases of Women, translated by Dr. Meadows, for New Sydenham Soc.
Aran, Leçons Clinique sur les Maladies de l'Uterus,
Courty, Traité pratique des Maladies de l'Uterus.
Virchow, Archiv. für path. Anat. und Phys., vol. xxiii.
Buhl and Hecker, Klinik der Geburtskunde.
Klob, Pathol. Anat. of Female Sexual Organs, Eng. Trans.
Kiwisch, Klinik Vorträge.
Scanzoni, Diseases of Females, American Trans.
Valleix, Guide du Méd. Pract. and Union Méd., 1853.
Matthews, Duncan, Peri-metritis and Para-metritis.
Thomas Gaillard, Diseases of Women, &c.
Becquerel, Maladies de l'Uterus.
Priestley, Edin. Month. Jour. Med. 1854.
Savage, Female Pelvic Organs.
Spencer Wells, Diseases of the Ovaries.
Neoggerath (Emil), Die Latente Gonorrhœa im Weiblichen Geschlecht.

INFLAMMATION OF THE OVARY.

BY JOHN WILLIAMS, M.D.

INFLAMMATION of the ovary occurring during the puerperal period will not be considered in this place.

Inflammation of the ovary in the unimpregnated female is not of infrequent occurrence. It rarely terminates fatally, and consequently opportunities seldom offer for the verification of the diagnosis or for observing the changes which take place in the organ in the course of the disease; yet the evidence obtained by clinical observation and by examination made after death leaves no doubt of the correctness of the general belief in the frequency of this affection. As the disease rarely proves fatal and the post-mortem proof of its existence has to be sought in subjects who have perished from other causes, and in whom the previous existence of ovaritis had not been suspected or inquired into, and whose menstrual history was not known, the only evidence of the frequency with which ovaritis occurs which can be expected to be obtained is the presence of fibrous bands or adhesions between the ovary and neighbouring organs, of thickening and opacity of the peritoneum covering the ovary, of the walls of the Graafian follicles, and induration of the ovarian stroma. Such conditions are by no means uncommonly met with. On account of the rarity of opportunities of examining the ovaries during an attack of inflammation, I will quote a description of an inflamed ovary found by Scanzoni in the body of a patient who died of pneumonia. He says: "The autopsy showed in the pelvis to the right of the uterus, a mass of coagulated fibrin of the size of the fist, easily separated from the adjacent organs, and evidently the result of an effusion. Having removed this, we came upon the ovary, which had a longitudinal diameter of 2·16 inches, a transverse diameter of 1·57, and a thickness of about 1·37 inches. Its form was oval, and it was considerably enlarged, as the measurements show. Its surface was of a violet-bluish colour, covered by numerous distended veins, and towards the inner angle of the posterior surface a recently ruptured follicle having a reddish-black colour was detected. The consistency of the organ was soft, in some parts almost fluctuating. On cutting into it there escaped a considerable amount of blood, and the surface of the section presented a violet colour and some greatly distended veins. The vesicle just alluded to as showing where a rupture had recently taken place, was about the size of a pea. It still retained in its centre a small quantity of black liquid blood, while its sides were coated by a tolerably thick lining of fibrin. Two neighbouring vesicles presented nearly the same measurements to the eye, and caused a slight protuberance on the surface of the ovary. On opening them, a fluid composed of blood and serum was poured out. Towards the other extremity of the ovary, where the congestion was less marked, the section less red, and the consistency firmer, an abscess was discovered in the parenchyma itself about the size of a bean, and containing pus mixed with blood. Behind this somewhat large abscess other smaller ones were discovered, whose size varied from that of a millet-seed up to that of a small pea. These were situated deeply in the parenchyma, and all contained more or less blood and pus. The entire tissue was infiltrated with serum, and most of the vesicles were perceptibly enlarged by a too copious accumulation of fluid."

In this instance the whole tissue of the organ was affected—the peritoneal covering, the Graafian follicles, and the stroma.

Four degrees of ovaritis have been described. In the first the organ is but slightly enlarged, its substance, though softer than natural, is firm, red, and injected, and the Graafian follicles increased in size. It is a state of congestion, and one that has repeatedly been observed, but only as a physiological condition in women who have died during or about a menstrual epoch. Though it must be admitted that congestion is, in the ovary as well as in other organs, the first stage of inflammation, yet owing to the difficulties with which the investigation of the anatomy of ovaritis is surrounded, and to the peculiar changes which take place in the organ in the performance of its functions, the first stage of ovaritis has never been observed in the dead subject. The second stage is that of red softening. The organ is enlarged to two or three times its usual volume; its tissue is soft and friable, is infiltrated with serum, and presents here and there small hæmorrhages. In the third, points of pus appear in place of the hæmorrhagic spots; these are usually found in the vesicles, but are also met with in the stroma. In the fourth degree softening and suppuration take place, and the ovary may be converted into an abscess, or break down into a sanious mass, as was observed by Kirvisch. The last stage is very rarely reached in the unimpregnated state. The second and third are analogous to the red and grey hepatisation of pneumonia, and have been observed in the same organ as was the case in the instance quoted from Scanzoni.

It is believed that each of the tissues of the ovary may be affected separately by inflammation, and three kinds of ovaritis have consequently been described—ovarian peritonitis, inflammation of the Graafian follicles or vesiculete of Negrier, and inflammation of the parenchyma. Ovarian peritonitis occurs frequently, as is proved by the adhesions found so often after death between the ovary and the neighbouring organs; whether the inflammation is limited to the superficial covering of the organ or extends for some depth into the stroma is uncertain. It usually extends to the peritoneum in the neighbourhood, though it does not become general.

Inflammation may attack one or more of the Graafian vesicles. The walls of the affected follicles are injected, red, and softened; their contents become turbid, bloody, or purulent. It may result in atrophy of the follicles, give rise to the formation of cysts, or run on to suppuration and abscess. When several follicles are affected, the intervening stroma becomes soft, œdematous, and friable: when suppuration takes place, the stroma between the several follicles breaks down and the ovary may thus become converted into a purulent sac.

Inflammation of the parenchyma is rare in the unimpregnated state. The ovary becomes enlarged, as already described. It occasionally ends in suppuration; more often it results in an excessive formation of fibrous tissue, which gives rise to permanent enlargement and induration of the organ, and atrophy of the follicles.

Abscess of the ovary may terminate in various ways. It may open into the peritoneum and cause death rapidly by shock, collapse, or general peritonitis; or a small quantity only of pus having escaped, may lead to circumscribed peritonitis and become encysted; or it may communicate with and discharge its contents into the bladder, rectum, or vagina; or, lastly, it may make its way through the abdominal walls and discharge on the surface.

Inflammation of the ovary is rarely, if ever, uncomplicated: there is present in addition inflammation of the Fallopian tubes and of the neighbouring peritoneum and organs. The acute form of the affection is very rare apart from childbed—so rare that many practitioners of the largest experience have not seen a single instance of the disease.

Its causes are exposure to cold during menstruation, excessive sexual indulgence, extension of inflammation from neighbouring organs—the uterus, Fallopian tubes, and broad ligaments,—gonorrhœa, operations on the uterus, the use of instruments, as intra-uterine stem pessaries, and intra-uterine injections: acute fevers.

Slavjansky states that inflammation of the follicles is not uncommon in the course of acute febrile diseases.

The form of ovarian inflammation usually met with, and which is very common, is chronic ovaritis. It may follow the acute or may arise independently of it; or it may result from the same causes which give rise to the acute form. By far the greater number of cases, however, may be referred to two classes—those which follow labour without apparently an acute stage, and those which are associated with dysmenorrhœa.

In the first class labour may have been perfectly normal and easy, or protracted and difficult; the woman apparently makes a good recovery, and suffers from no untoward symptoms until she leaves her bed. At that time or some time after a pain is felt in the region of one of the ovaries—usually the left; this may be at first slight, but is aggravated by walking or exertion. In the course of months it becomes more severe.

In the single, chronic ovaritis is in the very great majority of cases associated with dysmenorrhœa. Menstruation—which may be regular or irregular—is from the first associated with pain. The pain is in the back and pelvis, and continues to return at or about the menstrual epochs, ceasing during the intervals, for several years. After a long period a new and different pain is experienced; this is situated in the ovarian region and goes through to the back, and is due to ovaritis.

What may be the exact relation between the dysmenorrhœa and ovaritis is uncertain. It may be that the inflammation with which the inner surface of the uterus is affected in most of these cases, extends along the Fallopian tube until it ultimately reaches the ovary; or it may be that absorption of products of menstruation retained in the uterus takes place, and that the ovaritis is really septic in character. Whatever be the nature of it, the sequence of events mentioned is extremely common.

Syphilis is said to be a cause of ovaritis.

Phthisis and scrofula predispose to the disease.

The only case of abscess in the ovary seen by me was in a scrofulous patient. She died of acute peritonitis. Examination of the body showed the peritonitis to be due to bursting of a small abscess in the ovary. There was scrofulous disease of the kidneys.

The symptoms of the acute form are similar to those of pelvic cellulitis and peritonitis; and the intimate relation between these affections renders the diagnosis of ovaritis generally impossible. There is severe pain in the region of the affected ovary, some tympanitic swelling, and great tenderness on pressure, fever, and often a rigor. Examination by the vagina or rectum will discover the ovary enlarged, depressed, usually behind and on one side of the uterus, and exquisitely tender. If it be complicated with peritonitis, cellulitis, with effusion of lymph into the peritoneal cavity or with effusion into the cellular tissue, the enlarged ovary will not be detected. In rare cases the disease proves rapidly fatal by extension of the inflammation to the peritoneum generally, by suppuration and rupture of the abscess, or by rapid destruction of the ovarian tissue. On the other hand, resolution and recovery may take place, or the inflammation may become chronic.

In the treatment absolute rest is essential. The patient should be kept in bed. If seen early it may be advisable to apply ten or twelve leeches to the hypogastrium. Hot poultices or fomentations should be frequently applied to the lower abdomen, and opium should be freely administered.

In chronic ovaritis pain is felt in the region of the affected ovary. This radiates to the back and hips and down the thigh on the same side. It is of a dull or sharp character, and is at first periodic, lasting for a few days only at or about the menstrual epochs. After a time it becomes continuous, there are no intervals of freedom from suffering, and it is aggravated by walking and standing. There is often frequent micturition, and defæcation is in some instances exquisitely painful. Menstruation may be scanty, moderate in quantity, or profuse; sometimes there is amenorrhœa.

When the disease is of long duration, nervous symptoms set in. Curious sensations are felt in the side affected. These are not necessarily limited to any one spot, but are experienced over the whole of that half of the body. The patients become hysterical and often confirmed invalids. On examination of the abdomen one ovarian region is found fuller than normal—the fulness being due to flatus. Patients often say they have a swelling in that situation, and believe themselves to be the subjects of a tumour.

The part is tender, especially on deep pressure. Bimanual examination, made with one hand on the abdomen and a finger of the other in the vagina or rectum, will in most cases discover the ovary enlarged and tender. The organ is often but not always prolapsed, and occupies a position behind and a little to one side of the uterus. Pressure on it causes severe pain, and in some cases nausea. There is no marked fever, though there are often slight febrile attacks. The digestive functions are impaired. The tongue is furred, and the appetite lost; the bowels obstinately confined. Sterility is often present, especially in women who have not borne children, and who are the subjects of chronic ovaritis. Sterility is far less common in women who have acquired ovaritis after labour.

The disease very rarely proves fatal; it is, however, one of long duration. The chief danger lies in the liability to peritonitis. Both acute and chronic ovaritis are diseases of the reproductive period of life. The chronic form which follows labour is far more tractable than that associated with primary dysmenorrhœa. I do not think I have seen a case of the latter kind cured while the uterus and ovaries continued to perform their physiological functions.

Rest is an important element in treatment. Physical rest may be readily obtained, but physiological rest is not possible during menstrual life. Bromide of potassium is said to be useful, but I cannot say I have seen any good results from its administration, except when given in large doses at bedtime for three or four nights in succession before a menstrual epoch. Better results are obtained by general treatment and counter-irritation over the painful ovary. The bowels should be kept open by salines; sulphate of magnesia and soda are the best. They should be given three times a day, and the dose regulated so as to obtain one or two free actions daily. Purgation should be avoided. Blisters, or a liniment of iodine applied to the ovarian region, relieve the pain. The relief, however, is often only temporary. The production of an eschar or ulcer on the cervix of the uterus by the application of potassæ c. calce is said to be of service. In some cases relief is obtained by wearing a Hodge's pessary. This is doubtless due to the action of the pessary in keeping the ovary and uterus at rest. It will be found that the above means will effect a cure in a great many cases, more especially in those in whom the ovaritis can be traced back to labour; while in the majority, if not in all, of those in whom the ovaritis is dependent on primary dysmenorrhœa no means will be found to effect a cure, and the most we can hope to do is to arrest the course of the disease or ward off the onset of those nervous symptoms which mark it in its later stages and which render the patient a complete invalid.

OVARIAN TUMOURS.

By John Williams, M.D.

HE ovary is composed of two histological structures—an epithelium nd a vascular stroma. The first appearance of the organ, according o Waldeyer, is a thickened layer of germ epithelium, investing an utgrowth rich in cells projecting from the interstitial tissue of the Volffian body. By the increase of these two structures, and their eciprocal growth, the mass of the ovary is formed. In the course of evelopment the epithelium dips into the stroma, and processes of the atter grow into the epithelium. In this manner epithelial tubes and ngrowths of epithelium are formed in the stroma. The tubes are alled the tubes of Pflüger: they at first open on the surface. As the troma grows the tubes become closed, and the ingrowths of epithelium separated from the superficial layer and its processes. In this nanner isolated masses of epithelium become enclosed in the meshes f the ovarian stroma. These masses develop into Graafian follicles—he change being completed early in infancy. Foulis does not admit he existence of Pflüger's tubes and their development into Graafian follicles, but believes that the germ corpuscles which are embedded in the stroma become ova, that the Graafian follicles are the ultimate meshes of the stroma formed by the growth of the "connective tissue around the developing primordial ova," and that the "connective tissue corpuscles in the walls of the follicles in contact with the yelk develop into the corpuscles of the membrana granulosa." Whichever of these views be correct, we should naturally expect that the new growths formed in an organ possessing the structure described would often possess a cystic character; and experience does not disappoint the expectation, for by far the greater number of ovarian tumours are cystic.

Varieties.—Tumours of the ovary may be benign or malignant, solid or cystic. The solid and malignant are however rare.

Ovarian Cystic Disease.

Ovarian cystic disease is clinically divided into simple unilocular cysts, multiple cysts, multilocular cysts, and dermoid cysts. Pathologically a similar division holds—the two first being comprehended under dropsy of the Graafian follicle.

Dropsy of the Graafian follicle, hydrops folliculi, is the simplest form of ovarian cyst. It does not usually attain a large size. Such cysts are often not larger than a cherry; sometimes they attain the size of the fœtal head; and occasionally, but rarely, become so large as to fill the abdominal cavity and stretch the abdominal walls. When small they have a structure similar to that of the Graafian follicle; a fibrous coat derived from the stroma of the ovary—an inner coat on which the epithelial lining is placed, corresponding to the tunica propria of the follicle. Their contents are a clear fluid, and the ovum has in some instances been found in such cysts. As the cyst grows its walls become thicker and firmer, tougher, and more opaque. In thickness they vary much in different parts of the same cyst, as well as in different cysts. In some parts the wall may be extremely thin, while in others it may attain a thickness of one inch.

Cysts of this nature have been found in the ovaries of children,—even in those of the new-born,—as well as in those of adults. In the fœtus and child the condition is evidently due to hypersecretion of the fluid of the follicle, for at this period of life the Graafian vesicle contains little or no liquor folliculi. In the adult it may arise from the same cause. In many cases, however, it appears to be the result of some condition which prevents the rupture of the mature follicle and the escape of the ovum, such as thickening and induration of the coats of the follicle, in consequence of inflammation, and, it has been said, of congestion and hyperæmia.

Such cysts may moreover be developed from a ruptured Graafian follicle, or from a corpus luteum. Rokitansky says that cysts formed in this manner are "always lined by a stratum thicker than the wall of the follicle itself, which adheres to it either very loosely by delicate areolar tissue, or very intimately by a dense connective tissue. This lining stratum is of a dirty white colour, and has a rough inner surface. It may be recognised as the yellow layer of the corpus luteum which has been rendered thinner by expansion, and the roughness of its inner surface is occasioned by some of its remaining folds."

Several Graafian follicles in the same ovary may undergo cystic transformation, and form a *multiple cystic tumour*. The individual cysts of which such a tumour is formed possess a structure similar to that of simple cysts. As they grow and enlarge, one usually takes precedence of the others; they approach to and exercise mutual pressure on one another. In consequence of this pressure atrophy and absorption of the walls of the cysts takes place, and a communication is ultimately established between neighbouring cysts. As growth proceeds, the aperture of communication gradually enlarges, its borders retract and become less and less prominent, until finally two cysts completely merge into one, the only evidence remaining of the original condition being a slight ridge on the inner surface of the resultant cy or a slight thickening of its walls marking the line of union of the w of the original cysts. As this process affects the primary cysts in cession they gradually become merged int[illegible]mon cyst. I

OVARIAN TUMOURS.

By John Williams, M.D.

The ovary is composed of two histological structures—an epithelium and a vascular stroma. The first appearance of the organ, according to Waldeyer, is a thickened layer of germ epithelium, investing an outgrowth rich in cells projecting from the interstitial tissue of the Wolffian body. By the increase of these two structures, and their reciprocal growth, the mass of the ovary is formed. In the course of development the epithelium dips into the stroma, and processes of the latter grow into the epithelium. In this manner epithelial tubes and ingrowths of epithelium are formed in the stroma. The tubes are called the tubes of Pflüger: they at first open on the surface. As the stroma grows the tubes become closed, and the ingrowths of epithelium separated from the superficial layer and its processes. In this manner isolated masses of epithelium become enclosed in the meshes of the ovarian stroma. These masses develop into Graafian follicles—the change being completed early in infancy. Foulis does not admit the existence of Pflüger's tubes and their development into Graafian follicles, but believes that the germ corpuscles which are embedded in the stroma become ova, that the Graafian follicles are the ultimate meshes of the stroma formed by the growth of the " connective tissue around the developing primordial ova," and that the " connective tissue corpuscles in the walls of the follicles in contact with the yelk develop into the corpuscles of the membrana granulosa." Whichever of these views be correct, we should naturally expect that the new growths formed in an organ possessing the structure described would often possess a cystic character; and experience does not disappoint the expectation, for by far the greater number of ovarian tumours are cystic.

Varieties.—Tumours of the ovary may be benign or malignant, solid or cystic. The solid and malignant are however rare.

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Multilocular cysts, compound, composite, proliferous cysts, cystoma, cystoid, or adenoid tumours, have a very different structure. They are rarely single-chambered. In some instances they are formed of one large cyst, with a few small ones within it. In others the tumour forms a semi-solid mass, so divided into smaller cysts by partitions crossing its interior that it presents in section a honey-combed structure. Every transitional form, from the more simple to the most complex, may be met with. These tumours have been called colloid cysts on account of the character of their contents. Waldeyer calls them myxoid rather than colloid, because the contents of the ovarian cystoids are never pure colloid, and in order to express the true relation between the cysts under consideration and dermoid cysts: for he says that the inner surface of dermoid cysts has the character of epidermis, while the inner surface of myxoid cysts has the appearance and character of a glandular and vascular mucous membrane.

All cysts of the ovary are covered by peritoneum. This may be natural in character, or it may have become rough or adherent to neighbouring structures in consequence of inflammation. It has occasionally small villous, or globular epithelial growths on its surface. Waldeyer maintains that the ovary is not covered by peritoneum, and that this membrane does not form the superficial covering of ovarian cysts. He believes that the epithelium covering the ovary is not the homologue of that of the peritoneum, but that it is a columnar epithelium of a mucous character, having the same origin as, and precisely similar in character to, the epithelium lining the Fallopian tubes, with the one exception that the latter is furnished with cilia, while the former wants them. It is in this that he finds the cause of the frequent absence of adhesions between ovarian tumours and neighbouring organs. "Serous surfaces readily become adherent, and epithelial surfaces may become adherent to one another or to serous surfaces; yet in order to effect this the superficial epithelium must be destroyed. So long as the surface of an ovarian tumour is covered by epithelium it cannot grow to neighbouring structures, adhesions can only take place after the loss of the epithelium. When numerous adhesions are present the epithelium is always wanting." Waldeyer states that the villous excrescences on the external surface of ovarian tumours and the vegetations which protrude in some cases through the cyst wall do not form adhesions because they are covered by a well-marked columnar epithelium. Dr. Peaslee states that the part of the cyst wall formed by the ovary is not covered by epithelium, and that it can be distinguished by its greater whiteness and lesser degree of vascularity from the part of the cyst which is covered by peritoneum. Foulis however does not agree with the observations of Waldeyer upon this point. He states that the epithelium of the human ovary at six and twelve years of age consists of small flat hexagonal corpuscles, and regards it as homologous with the peritoneal epithelium.

The walls of the large cysts are separable into two layers, one external and one internal. The wall—especially the external stratum of it—of the principal cyst includes all the secondary or daughter cysts. It varies much in thickness. The external layer consists of tough fibrous tissue with very few cells; the internal layer is softer, more fleshy-looking and vascular, is composed of fine fibres with an abundance of cells. The walls are highly vascular; the veins especially are large, and are seen in great numbers under the peritoneum. The arteries lie deeper and penetrate to the inner surface, where they anastomose freely. They are of large size, and possess thick walls, and in some instances, according to Wilson Fox, retain the twisted or corkscrew-like appearance, which characterises those of the ovarian stroma.

The epithelial lining of ovarian cysts presents a variety of characters. It lines the whole of the internal surface, and forms usually a single layer of cells. According to Wilson Fox, the shape of the individual cells is usually a flattened polygonal approaching more or less to the circular form. In other cases they have a more flattened form, and are hardly distinguishable from the elongated cells of the connective tissue beneath. In other cases, according to the same authority, the epithelium assumes a stratified character, and forms several layers. In all the cases examined by Waldeyer it formed a single layer of cylindrical cells. Ciliated epithelium has been met with on the inner surface of cystoids by Virchow and others.

The walls of the smaller secondary cysts are formed of the inner layer only of the chief cyst wall. According to Waldeyer it is a tunica propria like that of the Graafian follicle in its early stage of development.

The mode of development of ovarian cystoids is still a subject of discussion. It was at one time thought that all ovarian cysts arose from cystic transformation of the Graafian follicle, and Dr. Wilson Fox, in an elaborate memoir published in the Transactions of the Medico-Chirurgical Society, has shown how the most complex cystic structures may arise from the Graafian follicle by the development of certain growths from its inner surface.

He describes three such growths—the papillary, cauliflower or dendritic, the villous, and the glandular.

The simplest forms of papillary growths are small club-shaped elevations on the inner wall of the cyst; they are at first composed of a hyaline finely striated tissue, with many elongated nuclei. They are covered by epithelium, and contain loops of vessels. These growths increase in size, and give off similar processes from their surface, and thus give rise to large composite masses. They are exceedingly vascular, their vessels being of a large size. They are solid, and now the central parts are fibrous, and the superficial hyaline, there being a gradual transition from the former to the latter. They may form over large portions of the cyst wall, or over small portions only. These growths give rise to formations of cysts in the following manner:—As the papillæ increase in size adjacent growths approach one another, and their surfaces come into contact at different parts. At the points of contact they unite, and inclose spaces lined by epithelium similar to that covering the papillæ and lining the principal cyst. These spaces are the commencement of secondary cysts. They may be found at the bases of the papillæ, or between the processes developed from them. They are of various and irregular shapes, but become round as their walls become distended by the accumulation of the secretion from their epithelial lining in their interior. The walls of such secondary cysts may develop papillary growths similar to those by means of which they were formed. These growths may form on their inner or outer surfaces, and may give rise to the formation of cysts in a manner similar to that already described, and these tertiary cysts may give rise to another formation of cysts by a similar process. Such multiplication or breeding may go on indefinitely.

Villi are very frequently found on the inner surface of ovarian cysts. They may be scattered over the surface or arranged in dense clusters. When scattered they do not attain a large size, and are often branched. They are covered by several layers of epithelium, which tends to assume the columnar form. When densely crowded together they give rise to the formation of tubular structures or cysts like the glands of the stomach. The first indication of the formation of villi is a stratification of the epithelium lining the cyst. Into these stratified masses of cells processes of the inner lining of the cyst containing a loop of vessels grow. In this manner a series of papillæ are formed with hollows or pits lined with epithelium between them, which are converted into tubular glands by a growth of the connective tissue of the stroma, between the papillæ and around the hollows. As the stroma grows upwards between the papillæ the tubular glands deepen and become more and more imbedded in the cyst wall. Vessels sprout up into the walls of the glandular tubules, and anastomose with those of the villi, forming arches at various depths of the tissue. These glands rarely multiply by lateral diverticula. A very common mode of increase, however, is by enlargement of the base of the gland, and a development of vascular papillæ from its centre in a fashion similar to the development of the primary villi. These papillæ may form tubular glands, and by a repetition of the process in the same gland its cavity may become subdivided by a series of septa into several tubes, all having a common outlet.

These glands may form cysts in various ways. The orifice of the gland may become closed by pressure on its sides; adhesion may take place between the touching surfaces, and the opening become sealed; or the gland may become dilated into a series of cysts, or follicles, across which septa may grow from the thickened walls and completely separate the several follicles, converting them into cysts; or the stroma of the ovary may grow upwards to such a degree as to include the whole of the papillæ, and inclose the whole of the gland in its substance. This would give an appearance of the development of glandular structures within the walls of the cysts. Indeed, in all cases in which the stroma of the wall has grown upwards between the papillæ, an appearance of the growth of glandular diverticula from the inner surface of the cysts into the substance of its wall is brought about. Dr. Wilson Fox, however, believes the above to be the true explanation of the process.

Dr. Fox describes another method in which multiplication of cysts may take place. The cysts produced in this manner are transparent, and possess very thin walls. The process is similar to that by which diverticula or buds are given off from glandular structures. Hollow processes, having a flask-shaped appearance, grow from the wall of the cyst. These processes have thin walls; their orifices of communication with the parent cyst are very fine and narrow; but their canals immediately expand, so as to form flask-like sacs. They are lined by polygonal epithelium, continuous with that lining the parent cyst. In this manner a multiplication of secondary cysts may go on indefinitely. Such secondary cysts, as they increase in size, may come in contact with one another, or with the walls of the parent cyst; union may take place at the points of contact, and several cysts may thus be merged into one, and leave no trace by which their mode of origin can be discovered.

Rindfleisch also believes that most ovarian cysts can be traced back to Graafian follicles, or to the tubes of Pflüger, and that the formation of secondary cysts results from the union of neighbouring papillæ developed within the primary cysts. Such union may take place in the course of, or at the extremities of, the papillæ. He maintains, however, that all ovarian cystomata do not arise from Graafian follicles, but that some are independent new formations in the stroma of the ovary. He says that "the connective tissue becomes in parts infiltrated with young round cells, and a colloid softening of the stromal connective tissue takes place:" and believes "that certain swollen transparent points in the stroma, which are not perfectly round and sharply defined, ought to be regarded as rudimentary cysts. In these the colloid matter is more diffusely infiltrated among the fibrous elements of the stroma; nevertheless, as it expands, it will inevitably tend to assume a spheroidal form, and to become marked off from the surrounding tissues as a globular cavity traversed by septa of connective tissue."

Waldeyer rejects both these views—that the Graafian follicle is the origin of cystoma, and that softening of portions of the stroma is the origin of cysts. He traces all cystoids of the ovary to the epithelial structures of the organ, and regards them as epithelial neoplasms. It has been already stated that according to this author, the Graafian follicles are developed from masses of the germ epithelium of the ovary, which have become embedded in the ovarian stroma. It is to these masses of epithelium that Waldeyer traces back the origin of cystomata. The transformation into Graafian follicles of these epithelial masses is completed, however, in the third year of life. Waldeyer accordingly maintains that a large number of ovarian cystoids date back their origin to a very early period of life, before the transition of the epithelial masses into Graafian follicles is completed, and that portions of the epithelial masses embedded in the ovarian stroma, instead of developing into Graafian follicles, undergo cystic transformation.

With regard to the origin of other cystomata, though no new follicular growths can be developed from the superficial ovarian epithelium after birth under normal circumstances, yet Waldeyer maintains that under pathological conditions such development is not only possible, but actually takes place. In proof of this he states that he has found immediately under the surface of the ovaries of aged women, small cysts up to the size of a pea, having clear, watery contents. They are like a cystoid in miniature, and are lined by columnar epithelium, which dips into the cyst wall in the form of tubular glands, corresponding to the glandular processes of a glandular cystoma. He has never found an ovum in such cysts, but he has in some instances found a direct connection between these cysts and the superficial epithelium of the ovary, and he regards them as the result of a pathological embedding of the superficial epithelium in the stroma of the organ.

Waldeyer thinks that the first development of ovarian cystomata does not start from the usual egg-containing Graafian follicle, but from a more embryonal form of the epithelial constituents of the ovary,—round epithelial or tubular structures, comparable to Pflüger's tubes, which originate probably from an embryonic period of the ovary, and were not developed into normal Graafian follicles, but took a pathological action from the first; or which originated from a repeated embedding, or ingrowth, from the surface of the ovarian epithelium.

Cystomata have generally been regarded as proliferous, that is they are formed of a parent or of parent cysts which have bred and formed secondary or daughter cysts, but Förster maintained that all the cysts forming a cystoma are formed side by side according to a primitive type. The view advanced by Hodgkin and Rokitansky, however, that they are proliferous, that secondary cysts form from parent cysts, and tertiary cysts from secondary cysts, etc., has been incontestably proven by the labours of Wilson Fox and others.

Waldeyer differs from Fox, not only with regard to the origin of cystoid, but also with regard to their mode of growth. Fox maintains, as we have seen, that there is here a peculiar mode of cyst formation, arising from the coalescence of the walls of neighbouring papillæ at various points of their course.

Waldeyer, on the other hand, denies this mode of cyst formation, and believes that the secondary cysts are formed in a manner similar to the development of cysts in other situations, that is by the formation of diverticula or sacs of the epithelium of the inner surface, which penetrate into the substance of the cyst wall. He figures such secondary cysts in the wall of the parent cyst, and one of his figures shows secondary cysts projecting on the surface of the parent cyst, the secondary cysts being formed apparently by the dilatation of glands, in consequence of the accumulation of their secretions, from the plugging of their orifices by gelatinous contents. There does not appear to be anything in these, however, inconsistent with the view of Wilson Fox.

Cystomata may occur in one or both ovaries, and form tumours of various magnitudes. They are often so large as to fill the abdominal cavity, and distend the abdominal walls, causing œdema of the parietes and of the lower limbs. Some have a tendency to become unilocular, while others become more complex and multilocular. Peaslee has accordingly named the former oligocystic and the latter polycystic. It is said that the younger the tumour the greater generally is the number of cysts, while old tumours may be formed of one large cyst with few or no small ones.

Ovarian cysts are attached to the uterus by a pedicle. This may be short or long, stout or slender; sometimes it is tough and firm, in other instances it is fragile and breaks down easily, hardly bearing a ligature. It is composed of the ovarian ligament, the Fallopian tube, and the broad ligament. The extremity of the Fallopian tube is often adherent to the tumour, its fimbriæ being spread upon it. At the point of attachment of the tube, or near to it, are to be found the remains of the ovary, should any portion of the organ be not implicated in the growth. In the pedicle the blood vessels, which are sometimes very large, and the lymphatics, run into the cyst.

The contents of ovarian cysts are extremely various. They may consist of a thin clear fluid having a specific gravity of 1,007 to 1,015, or of a firm jelly; while they may present every degree of transition between these two forms. Usually the larger cysts have the more fluid contents, and the smaller the more solid. This is true of the several cysts entering into the formation of the same tumour as well as of cysts from different tumours. The fluid may be pale, clear, and transparent. This is especially the case in unilocular cysts. When such cysts have been tapped their contents become thicker, more viscid, and of a darker colour; occasionally they resemble an infusion of coffee, sometimes they are blood-stained. Contents of a green, yellow, dark brown, black, inky, like gruel or custard, and a mixture of fluids or solids like brain matter have been met with.

The amount varies according to the size of the tumour. Forty or fifty pounds of fluid is not uncommon, and 130 or even 160 has been reached. When tapping has been had recourse to the character of the fluid withdrawn is altered. It usually becomes thicker and darker with each successive operation.

Chemically, according to Eichwald, the contents of cystomata may be divided into two classes, the mucous and the albuminous.

The mucous series consists of—

The material of colloid globules,
Mucin,
Colloid material, and
Mucous peptone.

These four substances are distinguished only by their different degree of solubility in various liquids.

The material of colloid globules dissolves in weak alkalies.

Mucin dissolves in solutions of the alkaline earths, and swells in an extraordinary manner in water.

Colloid matter is partially readily soluble in cold, and still more so in hot water.

Mucous peptone dissolves readily and completely in water at any temperature.

In the same degree as these substances require alkalies for their solution will they be precipitated from their solutions by acids; so that the material of colloid globules and mucin are completely, colloid material incompletely, and muco-peptone not at all precipitated by acids. The precipitate is insoluble in excess of acetic acid. On the other hand every member of the group is soluble in mineral acids, but not in the same degree; the material of colloid globules being the least and muco-peptone the most readily dissolved by these solvents.

The substances are never precipitated from their acid solutions by ferro-cyanide of potassium.

They are not usually precipitated by tannin, or by neutral metal salts, but are completely thrown down by basic lead salts.

The albumen series consist also of four substances—

Albumen (and fibrine),
Paralbumen,
Metalbumen,
Albumen peptone (fibrine peptone).

These are distinguished from the mucin series generally by their being precipitated from their solutions by tannin and neutral metal salts. The first three moreover undoubtedly contain sulphur. There is doubt whether this element enters into the formation of albumen peptone.

As regards the solubility of this group, a gradation similar to that found in the mucin series is met with. Like mucin, albumen is insoluble in water, dissolves however in alkalies, and is precipitated from its solution by acids. Albumen peptone, like mucous peptone, is completely soluble in water, alkalies, and acids. Paralbumen and metalbumen correspond to colloid material. Paralbumen appears to be more soluble than albumen, as influences which throw down the latter by the withdrawal of water from animal fluids, precipitate the former less readily and completely. A further distinction lies in the fact that the combinations which paralbumen forms with mineral acids are more readily soluble in water than the corresponding combinations of albumen.

The solids usually found on microscopic examination are granules, globules of fat, granular cells, epithelial cells, crystals of cholesterine, blood corpuscles, and disintegrated blood, pus cells and compound granular cells or inflammatory globules of Gluge. Some of these are present in the contents of every ovarian cyst, but it is rarely that all are found together in one specimen. Of these bodies the most important is the granular cell, and it is almost invariably present.

According to Dr. T. M. Drysdale, "This granular cell, in ovarian fluid, is generally round, but sometimes a little oval in form, is very delicate, transparent, and contains a number of fine granules, but no nucleus. The granules have a clear, well-defined outline. These cells differ greatly in size, but have always the same structure. They may be seen as small as $\frac{1}{25000}$ inch in diameter, and from this to $\frac{1}{2000}$ inch. In some instances I have found them much larger, but the size most commonly met with is about that of a pus cell. The addition of acetic acid causes the granules to become more distinct, while the cell becomes more transparent. When ether is added the granules become nearly transparent, but the appearance of the cell is not changed. This granular cell may be distinguished from the pus cell, lymph corpuscle, white blood-cell, and other cells which resemble them, both by the appearance of the cell and by its behaviour with acetic acid."

He adds, "This ovarian granular cell I consider as diagnostic of ovarian dropsy, and have seldom failed to find it in this fluid, except in some of the earlier cases, where it probably existed but was overlooked from inexperience in the examination of these fluids."

Dermoid cysts though occurring most frequently in the ovary, have been met with in other regions, and in the male as well as in the female. Lebert applied to them the term dermoid because they contain skin.

The wall consists of two layers, an internal and an external.

The inner coat is smooth, having prominences in different parts; it consists of true skin. The epithelium consists of several layers of cells, the innermost of which are flattened and non-nucleated, while the deeper are round or polygonal in shape. Under the epithelium is a layer of tissue similar to the cutis, frequently having papillæ on its surface, but not always. When papillæ are present they are arranged irregularly, and do not run in rows like those seen on the skin of the palm or fingers.

Beneath this cuticular layer is a stratum of fat, analogous to the panniculus adiposus. This is inclosed in an external fibrous envelope.

The layer of the cutaneous wall contains hair follicles with which sebaceous glands are connected. Other sebaceous glands open on the surface. Heschl and others state that they have found sweat glands also in this wall.

The contents of dermoid cysts consist of some fluid, and a greasy yellowish substance composed of epithelial cells and sebaceous secretion. Sometimes cholesterine is present in great abundance, and traces of oxalic acid are said to have been found.

Hair, teeth, and sometimes bone, brain substance, nerves and muscular fibre form part of the contents of these extraordinary structures.

The hair formed in them is usually fine like wool, of a blonde or reddish colour, but sometimes dark. It varies in length from a few inches to two feet. It is generally rolled up into balls and separate elongated masses, which are sometimes completely isolated from the inner surface of the cyst by being lodged in a capsule. In some instances long hairs have been found attached to the inner surface.

Perfect teeth having the characters of incisors, canines, molars, have been met with in dermoid cysts, and in some instances they have been found to follow the order of succession occurring naturally; that is a deciduous tooth has been found to undergo atrophy and absorption, in consequence of the pressure upon its fang caused by a permanent tooth growing under it. Such perfect structures are however of rare occurrence, and usually the teeth found in these cysts are rudimentary or imperfect. Many are badly formed, either from excess or deficiency of growth. They arise from bone or from the stroma of the cyst wall. The number present varies. As many as three hundred have been found in a single cyst. Commonly there are few—1 to 10 present.

The osseous structures occur in the form of laminæ, or masses of irregular shape, firmly imbedded in the cutis. They are usually small, but pieces as large as the palm of the hand have been met with. They have the true structure of bone, but the laminæ are larger and fewer than those of bone, and the canaliculi are less numerous. Instances have also been recovered in which pieces of bone have been articulated by loose capsular ligaments. Vanderwich found a small, hollow bone, with an external covering like periosteum, and lined by a membrane analogous to the dura mater.

Striped muscular fibres have been discovered in dermoid cysts by Virchow, and brain substance and nerve chords have several times been detected.

Dermoid cysts have been regarded as the result of the imperfect development of an ovum, taking place either spontaneously or in consequence of impregnation. Their origin has also been explained by the early inclusion of an ovum which is imperfectly developed within another ovum which attains perfection. On the latter supposition two ova become impregnated at about the same time, and in the course of development one becomes inclosed in the other; the latter undergoes full development, while the former develops skin, hair, and bones only.

The evidence, however, against all theories which refer the origin of dermoid cysts to the development of an ovum under any circumstances is overwhelming.

They have been seen in organs other than the organs of generation, as the lungs, thyroid glands, kidneys, &c. They are not infrequently observed before puberty, when the influence of the male was not possible: they have been met with before birth.

With regard to the theory which refers their origin to the inclusion of one ovum within another, the above objections do not apply, and yet this theory is no less unsatisfactory than those which refer their origin to the development of a single ovum. That inclusion of a germ within a germ does occasionally take place is not improbable, and this fact would render the theory at first plausible.

About three-fifths of dermoid cysts are found in the female, and two-fifths in the male: and three-fifths of all dermoid cysts are found in the ovary, while the majority of those met with in the male are in the testicle: so that there is some cause which determines the formation of these growths in the generative organs of the male and the female, and this renders highly improbable the theory of inclusion.

The view of the origin of these cysts now generally received is that they are congenital and due to a displacement of the external layer of the blastoderm. From this layer the epidermis and other structures are developed, and it is supposed that a portion of it becomes included in the part of the middle layer from which the ovary is formed, and forms the rudiments of cysts of a dermoid character.

Tubo-ovarian is a term applied to those cysts whose walls are in part formed by the ovary and in part by the Fallopian tube. The portion of the cyst formed by the ovary is lined by a smooth or uneven yellowish membrane, which is not continued into the part formed by the tube. A part only or the whole of the Fallopian tube may contribute to the formation of such cysts. The junction of the tube and ovary is marked occasionally by a slight constriction. The greater part of the cyst is formed by the ovary. Such cysts occasionally pour their contents into the uterus along the Fallopian tubes and become collapsed. Mr. Spencer Wells has described such cases, and has in some instances found the collapsed cysts on examination after death.

Tubo-ovarian cysts are said to be formed in the following manner:—At the time of rupture of a Graafian follicle the point of rupture is grasped by the fimbriæ of the Fallopian tube. After the rupture has taken place the fimbriæ do not retract, but remain adherent to the ovary. The corpus luteum resulting from the ruptured follicle undergoes cystic degeneration, as is shown by the yellowish lining of the ovarian portion of the cyst, the communication between the follicle and the canal of the tube is maintained, and in this manner the cyst is formed jointly by the two structures.

Cysts may moreover be developed in the immediate neighbourhood of the ovaries, in the Fallopian tubes, and the broad ligament.

Cysts of the Fallopian tubes do not usually attain a great size, but Dr. Peaslee relates a case in which a cyst having this origin had attained a capacity of eighteen pounds. They are the result of inflammation of the tube causing closure of its orifices or obliterating it in some parts of its course, and of an accumulation of secretion or of an effusion of blood into its canal. The outer being the most distensible part of the tube the principal enlargement occurs at that part, and the rest remains less distended, maintains a tortuous course, and is sometimes sacculated in consequence of constriction by inflammatory products. Both tubes are usually affected; the wall is thin, the lining membrane smooth or rough, and exhibiting papillary growths.

The contents may be watery, mucous, sanguineous, or purulent.

Cysts of the broad ligaments are of two kinds, those arising from the terminal vesicles of the Fallopian tubes, and those arising from the tubules of the par-ovarium. The cysts arising from the terminal vesicles of the Fallopian tube or remains of the Wolffian body are usually small, about the size of a pea or a nut; occasionally they attain the size of an egg. They are covered by peritoneum, have very thin walls, clear watery contents, and hang by a slender pedicle. They often burst, and their contents escape into the cavity of the peritoneum, but give rise to no trouble. They have been discovered not so often during life as during post-mortem examination.

Cysts derived from the tubules of the par-ovarian occasionally grow to a large size. They are covered by peritoneum; their wall is composed of fibrous tissue, but it is comparatively thin, and is lined by columnar epithelium. Their contents consist of a thin, clear, serous fluid. They rarely refill after tapping, or after accidental rupture of their walls.

CAUSES.—Very little is known concerning the causes of ovarian cysts. This is perhaps due in part to the fact that it is not possible to fix the exact date of the commencement of the disease. The first evidence of its presence is an abdominal tumour, but at that time the disease has lasted some considerable time. There may be nothing to note in the previous history, or there may have been pain in the region of the diseased ovary, and general failure of the health; such data are not enough to fix the time of the commencement of the disease.

No age is absolutely free from it. As far as observation at the bedside goes, it is very rare before puberty; it has, however, been observed in children of all ages. On the other hand, observation after death shows that the disease is by no means uncommon at birth. Small cysts are frequently met with in the tissues of the fœtus; and some have maintained that the majority of all cystomata as well as dermoid cysts have a congenital origin. If they be of congenital origin, it is after puberty they usually begin to grow—during the period of functional activity of the organs of generation. Nearly a half of the cases occur between thirty and forty years of age. They are met with after the menopause and occasionally at a late age.

The exercise of the sexual functions does not predispose to ovarian cystic disease. West found that of 327 cases 94 were single, 31 were widows, and 202 were married or had had children; that is "considerably more than a third of all cases of ovarian disease began at a time when the sexual functions were not in active exercise; and more than a fourth occurred in women in whom these functions had never been exercised at all."

Sanzoni regards chlorosis and disorders of the uterine functions as predisposing causes of ovarian cystic disease; while others have not found such a relation between the two affections. It is however certain that women the subjects of ovarian cysts are less prolific than others; while many of them are absolutely sterile. The relation between the two conditions is uncertain; it may be that sterility and a less degree of fecundity favour the development of ovarian cysts; while, on the other hand, and this is the more probable, it may be that in most cases of this disease there is from the first the abnormal condition of the ovaries which accounts for the absolute or comparative sterility present, and which tends to develop cysts. The tendency of modern pathological research is in favour of the latter view.

COURSE.—Ovarian cystomata tend to grow until they fill the abdominal cavity, distend its walls, encroach upon the thoracic cavity, press up the abdominal and thoracic viscera, and finally cause death by interfering with the nutrition of the body and the functions of the vital organs. They are, however, subject to a variety of changes which may affect their course in a marked manner. These we shall briefly notice.

Cases of spontaneous absorption of ovarian cysts and cure have been recorded. There is no doubt that a considerable quantity of fluid supposed to have been inclosed in a cyst of the ovary has in some instances disappeared and this has been accompanied by a profuse discharge of a watery fluid from the bladder or bowel. It is generally believed that so long as the fluid is confined in the cyst it is incapable of absorption, and the process which takes place in the alleged cases of absorption is somewhat uncertain. It may be, as Barnes suggests, that the fluid had first escaped into the peritoneum, and became then absorbed, or into one of the neighbouring organs in consequence of a small perforation.

In some instances the growth of the tumour has ceased suddenly and finally: in other cases the quiescent state has been temporary only. Ritchie has recorded a case in which increase and diminution in the size of an ovarian tumour alternated until it terminated fatally. Cure has resulted from rupture of the cyst, the discharge of its contents into the peritoneal cavity, and their subsequent absorption. In most cases where the contents escape, whether by tapping or injury, they reaccumulate. Rupture may result from violence, blows, or falls, or spontaneously without assignable cause.

The escape of the contents of an ovarian cyst into the peritoneal cavity is rarely free from danger; it frequently proves fatal. When the fluid is of a bland and non-irritating character it becomes readily and rapidly absorbed when discharged into the cavity, and is carried off by the skin or kidneys. Should it be thick and irritating, as the contents of polycysts frequently are, severe and fatal inflammation is almost sure to follow.

Cysts sometimes become the seat of inflammation, either spontaneously or in consequence of accident or injury. This may lead to the formation of pus or of a foul and offensive fluid in the cavity of the cyst, or to general peritonitis. The offensive fluid may become absorbed and give rise to pyæmia or septicæmia and death: or the cyst may become perforated, the foul matter be discharged into the peritoneal cavity, and set up rapidly fatal peritonitis.

In some instances the pedicle has become twisted. In such cases the blood vessels are constricted; there is obstruction to the return of the blood from the tumour. This may lead to gangrene or hæmorrhage into the cyst, causing sudden distention of the abdomen, collapse and death. Should the twisting be brought about in a gradual manner, the pedicle may undergo gradual atrophy, and the tumour be ultimately separated from its attachment and lie free in the cavity of the abdomen.

The contents of an ovarian cyst may be discharged into neighbouring organs. Adhesions form between the cysts and the organ, and ulcerative perforation or rupture of the cyst wall takes place at the place of union. In this way the cyst contents may be discharged into the Fallopian tubes, the bladder, bowel, vagina, or into the lung and pleura. When an opening forms from the intestine into the cyst, the contents of the bowel may enter its cavity and give rise to fæcal abscess and death.

In some instances great hæmorrhage has taken place within an ovarian cyst, without assignable cause.

The pedicle may give way spontaneously, and the tumour float in the abdominal cavity, or become attached to some other part and continue to grow.

Death occasionally takes place from asphyxia, arising from pressure on the heart and lungs; in other rare cases sudden and fatal collapse has occurred.

SYMPTOMS.—The symptoms of ovarian cystic tumours vary much. Different persons manifest tolerance of such growths in very different degrees. In some cases no symptoms are apparent until the tumour has attained a size sufficient to cause a distinct swelling above the pubes. In other cases, troubles of a more or less severe character are present from an early period. Pain in the back and a sense of weight in the pelvis are commonly complained of during the early stages—while the tumour is small and lodged in the pelvis. Occasionally severe pain on one side, in the ovarian region, is present before any signs of tumour are manifest. Menstruation may be performed regularly and normally, or it may be accompanied by pain, or amenorrhœa or menorrhagia may be present. The presence of the tumour in the pelvis causes usually a certain amount of trouble in the performance of the functions of the bladder and of the rectum. Irritation of the bladder or frequent desire to micturate is often present; occasionally there is incontinence and now and then retention of urine from pressure of the tumour on the neck of the bladder or the canal of the urethra. Irritation of the rectum, constipation, and hæmorrhoids are frequent associates of ovarian growths. Sometimes pains are felt in the lower limbs, owing to pressure on the nerves as they pass through the pelvis. Occasionally symptoms which are usually referred to pregnancy appear during the early stages of ovarian tumour, such as morning sickness, swelling of the breasts, enlargement and discolouration of the nipples and areola, and these may run a course precisely similar to that which they run usually during gestation. I have seen one case where the appearances of the breast were such as are commonly said to be diagnostic of pregnancy: the nipple was prominent, the areola darkened, enlarged, and shining, the follicles swollen, and the so-called secondary areola perfect. A milky fluid may also appear in the breasts.

In some cases the general health suffers from the first or from an early period. The digestion may be impaired and the appetite lost, and a feeling of sickness be constantly present; there may be a general sense of illness or even decided suffering. When the tumour has attained so great a size that it cannot be contained in the pelvis, and that it rises into the abdomen, other symptoms make their appearance. Sometimes the growth remains imprisoned in the true pelvis on account of adhesion, or for some other reason, and then the rectal and vesical symptoms become aggravated and pain becomes severe. In such cases the bladder suffers more than the rectum by reason of the protection given to the rectum by its position in the pelvis. When the growth ascends above the pubes it forms a distinct tumour on one side of the middle line which can usually be distinctly felt on palpation. It often causes some pain, though itself is not tender unless it or its peritoneal covering be the seat of inflammation. As it grows it becomes less lateral in position, and when it has reached the umbilicus it usually distends the abdomen in an almost uniform manner. The symptoms at this time are chiefly and usually due to pressure. There is a sense of fulness, weight and pain in the back. There may be œdema of one leg and pain, owing to pressure on the veins and nerves. At a later period both legs suffer. The abdomen becomes larger, the superficial veins in the abdominal walls become enlarged, owing to obstruction to return of blood along the common iliac veins; lineæ albicantes appear by reason of the stretching of the abdominal walls; the intestines and stomach become pressed upon and their functions interfered with; constipation, disorders of digestion and vomiting set in; the general health begins to suffer, and in consequence of this amenorrhœa follows. Later still the thoracic organs become the subjects of pressure: the diaphragm and heart are pushed up, the lower ribs spread out, the bases of the lungs pressed upon, so that palpitation, dyspnœa, and imperfect aëration of the blood result. The circulation in the kidneys becomes interfered with, and albumen appears in the urine. Emaciation sets in and this is of a peculiar character, as it chiefly affects the face, neck, and chest, and upper limbs. It gives rise to the so-called facies uterina or ovariana. Mr. Spencer Wells says, "the emaciation, the prominent or almost uncovered muscles and bones, the expression of anxiety and suffering, the furrowed forehead, the sunken eyes, the open, sharply-defined nostrils, the long compressed lips, the depressed angles of the mouth, and the deep wrinkles curving round these angles, form together a face which is strikingly characteristic."

The further progress of the disease leads to aggravation of the above symptoms, and death in one of the various ways already mentioned.

DIAGNOSIS.—The diagnosis of ovarian tumours is often very easy, sometimes difficult, occasionally not possible. The abdomen has been opened in several instances, with a view to the removal of an ovarian cyst when not only no ovarian but no other tumour was present. Before entering upon a discussion of the symptoms by which ovarian growths can be distinguished from other abdominal or pelvic tumours, it is necessary therefore to exclude those conditions which may give rise to enlargement of the abdomen, and which may either simulate new growths, or may lead the patient to suppose herself the subject of such growths. These are tympanites, obesity, œdema of the abdominal walls, phantom tumour, and spurious pregnancy.

Great and persistent tympanites occurs in the subjects of chronic dyspepsia. There is often great distress, with uncontrollable belching. The abdomen is tense, uniformly enlarged, and everywhere tympanitic on percussion. The last symptom distinguishes it from solid or cystic ovarian tumour.

Towards the menopause, and in some instances at other ages, women rapidly deposit fat in the abdominal walls and mesentery or omentum. In these cases the layers of subcutaneous fat can be grasped by the hand and lifted, and the umbilicus is hollow and depressed. If there be great deposit in the omentum an indistinct tumour may be felt. On percussion, however, the note is not absolutely dull, though it may not be resonant; the hand may be made by firm and prolonged pressure to sink to the spine and the sensation derived from the mass is doughy and not firm and resistant.

Phantom tumour is seen in hysterical women: it is formed often involuntarily, but in many cases at will. It appears to be due to the contraction of the recti muscles. By careful palpation when the attention of the patient is engaged in conversation or otherwise, or during deep inspiration, the hand may be pressed down to the spine and the absence of a real tumour ascertained. If any doubt remain, an anæsthetic should be administered, when the tumour, if of this nature, will entirely disappear.

So-called spurious pregnancy may occur at almost any age, but most usually it occurs about the menopause; that is, women may, for some reason or no reason, imagine themselves pregnant when they are not, and suffer from many of the symptoms which are present during pregnancy, such as morning vomiting, arrest of menstruation, and so-called quickening. In these cases there is no enlargement of the belly unless due to obesity or tympanites. No tumour can be discovered on examination and the uterus is of normal size.

Œdema of the abdominal walls is readily detected by putting on pressure; but this is a condition which not infrequently accompanies large ovarian tumours, and though it has been mistaken for ovarian growths, yet ordinary care will distinguish between the two conditions.

When the above conditions have been excluded, and it has been ascertained that the patient is the subject of an abdominal tumour, it remains still to decide whether it has an ovarian or other origin. There are many conditions which are liable to be mistaken for ovarian growths, and none of them are of greater importance in practice than enlargements of the uterus.

Pregnancy is the most common form of uterine enlargement. In many cases it is readily distinguished from ovarian cyst, yet instances have occurred in which the pregnant uterus has been mistaken for ovarian tumour; and in some cases the diagnosis presents great difficulty. The history is of some service in all cases in which it can be obtained; in some cases, however, the physician is misled by the patient, either accidentally or intentionally, and he has to fall back upon and trust to the results of physical examination alone. Setting aside then the history of menstruation and sexual intercourse, the breasts should be examined. Should they be enlarged, the nipple prominent, with darkened areola and enlarged glands, pregnancy may be suspected, for though these signs may occur with ovarian tumour, they occur with it exceptionally only. During the first two months of pregnancy the uterus remains in the cavity of the pelvis, and bimanual examination cannot fail in ordinary circumstances of recognising the enlargement of the body of the organ. In the third month the uterus rises and can be felt in the pelvic brim. It does not give a sense of fluctuation until the fifth month. Before that period it feels like a softish, solid mass. After the fifth month fluctuation is present until the full term. This may vary much in degree. When the liquor amnii is abundant, fluctuation may be very distinct, when scanty, difficult to recognise. In both conditions, however, a solid body, the fœtus, may be felt floating in the fluid when the latter is displaced by pressure. During the latter months of pregnancy it is possible in many cases to trace the outlines of the fœtus, and it can be pushed from side to side and made to alter its position. An ovarian tumour complicated with ascites or occurring in a subject having thick abdominal walls, may be mistaken for a fœtus; but when the spontaneous movements of the fœtus are distinctly felt, they are conclusive of pregnancy. Care should be taken that the movements of the abdominal muscles and of the intestines be not mistaken for those of the fœtus, which in some cases they greatly resemble.

In the fifth month the sounds of the fœtal heart become audible: these are diagnostic of pregnancy. A sound similar to the placental murmur is often heard in fibroid tumours of the uterus, and occasionally, though rarely, in ovarian growths, so that this sign is of no great value in diagnosis. The changes in the cervix of the uterus which are found in pregnancy do not occur in ovarian tumours. Hence Wells says that the changes in colour of the mucous membrane of the vagina seen during pregnancy are not seen in ovarian cystoma. Peaslee, however, states that this sign is of no value, and attaches greater value to the umbilical areola. The duration of a case often settles the diagnosis, especially when in conjunction with it the size of the tumour is taken into consideration. In extra uterine pregnancy, which is rare, there is during the early period of the disease, the history of pregnancy, and vaginal examination discloses a soft uterus and cervix. Other signs of pregnancy soon make their appearance, should the case go on; on the other hand, should the cyst rupture, the condition present becomes known or suspected. In molar pregnancy there are usually some signs directing attention to the uterus rather than to the ovary, such as hæmorrhages, and watery discharges; and in some cases portions of the contents of the uterus escape by the vagina. The diagnosis of ovarian cyst complicated with pregnancy is very difficult because "the enlarged uterus remains in front of the cyst, and no change of posture brings the latter into a situation where it can be reached and fluctuation be detected in it." When during pregnancy the uterus is situated high up so that the cervix is beyond the reach of the finger, and when the abdomen presents an uneven surface, the complications in question may be suspected. In many of the cases in which the condition has been met with, the presence of an ovarian tumour had been recognised before impregnation occurred, while in others the complication was not suspected until it was discovered during the performance of ovariotomy.

There are other enlargements of the uterus which may be mistaken for ovarian tumour. Of these, fibroid and fibro-cystic tumours present the greatest difficulty in diagnosis. In the very great majority of cases the diagnosis is readily made, while in others it is impossible. The following points of difference I abstract from Mr. Spencer Wells' work on Diseases of the Ovaries.

The enlargement of the abdomen is more often general in ovarian and partial and confined to the lower part of the belly in uterine tumour. The umbilical depression is diminished, or the umbilicus may become prominent, in large ovarian cysts. This is rarely seen in uterine tumours unless ascites be also present. The superficial veins of the abdomen are oftener found enlarged with uterine, though not uncommon with large ovarian tumours. Peaslee, however, states that the abdominal veins are not enlarged in uterine tumours. Both kinds of tumour move with respiration, but uterine tumours appear fixed at their lowest part in the hypogastrium.

When the recti muscles are called into action they "bulge forward in front of a tense non-adherent ovarian tumour or a flaccid adherent cyst. This is seldom well marked in uterine tumours, a solid mass fixed centrally below the umbilicus interfering with the free action of the recti. The girth of the abdomen is increased more on one side than on the other ovarian, more often symmetrically in uterine tumours. The increase of the distance between the pubes and umbilicus is proportionately greater in uterine than ovarian growths. If the tumour be cystic it is presumably ovarian, if solid presumably uterine. Fibro-cystic tumours of the uterus and solid tumours of the ovary are occasionally, though rarely, met with. Uterine tumours are less moveable than ovarian, unless the former be pedunculated; the ovarian tumour may be pushed up or raised from the pubes so as to permit the hand to be pressed down between it and the bone; but a uterine tumour cannot be raised at all, and the hand cannot be pressed between it and the pubes as pointed out by McClintock. Both ovarian and uterine tumours give dull notes on percussion, and a murmur may often be audible over uterine tumours, but very rarely over ovarian growths.

By vaginal examination the size and character of the uterus and its relation to the tumour may be determined. If the cervix is normal and the uterus is not enlarged, the tumour is probably not uterine. On the other hand if the uterus be heavy, the cervix and body hard and thickened, the canal distorted and elongated, a condition which may be detected by the uterine sound, then it is almost certain that the tumour has a uterine origin. It should, however, be borne in mind that ovarian tumours may displace the uterus backwards, upwards, downwards, forwards, or laterally, may distort its shape and elongate its cavity; and on the other hand that subperitoneal pedunculated tumours of uterine origin may grow without giving rise to any change in the position and characters of the uterus, and may permit the womb to be moved quite independently of them.

Examination by the rectum is often useful. If the whole hand be introduced into the bowel the size of the uterus, its position, and its relation to the new growth may be accurately determined.

Ovarian tumour and uterine tumour are sometimes present in the same subject. By keeping in mind the possibility of the occurrence of such a complication a diagnosis may be arrived at from a consideration of the symptoms just stated.

From other enlargements of the uterus—hæmatometra, hydrometra, and physometra—ovarian tumours may be readily distinguished.

Hæmatometra forms a median fluctuating tumour which increases gradually and is associated with amenorrhœa and severe pains at the monthly epochs. If the cause of it be congenital, the patient has never menstruated; if acquired, it has been the result of accidents during labour. Local examination will discover closure of the genital canal. Hydrometra occurs in aged women after the menopause, and is due to an accumulation of secretions in the uterine cavity in consequence of closure of the canal of the cervix. Both hydrometra and hæmatometra are rare conditions.

Physometra or accumulation of gas in the uterus is very rare. It occurs in hysterical women, and is accompanied often by escape of gas by the vagina. Percussion would give a tympanitic note, and thus distinguish it from ovarian cyst.

In the majority of cases, and where the abdominal enlargement is not excessive, there is no difficulty in distinguishing between ascites and ovarian cyst. When the abdominal distension is very great, however, much difficulty may be experienced. In ascites there is a history of previous illness, of disease of heart, lung, liver, or kidneys, or of peritonitis. There is œdema of the legs in many cases at an early period, a puffiness, and a pallid appearance of the face. The swelling increases more rapidly than in ovarian disease.

When the patient is lying on the back the shape of the abdomen is different: it is uniformly enlarged, flattened in front, and bulging in the flanks. Uniform enlargement of abdomen is present only in the last stages of ovarian cyst, and the front of the abdomen is always prominent. In ascites, when the patient sits up, the lower part of the abdomen bulges. When lying on the back, there is dulness on percussion in the flanks and depending parts of the belly, but not in front and the most elevated parts. The space over which dulness is found depends upon the amount of fluid present and the amount of air in the intestines. Fluctuation is felt in the dull parts. The seat and area of dulness and fluctuation are altered by position. The fluid gravitates into the most depending parts, and it is there that these signs are met with; so that when the patient is lying on the side, the lower flank is dull and the upper resonant; when she is in sitting posture, the lower part of the abdomen is dull and fluctuating and the upper resonant. In ovarian dropsy the most prominent part of the tumour is dull and fluctuating, and the area of dulness and fluctuation is not altered by change of position, and the flanks are resonant.

When the accumulation of fluid in the peritoneum is very great, however, the abdomen may be everywhere dull and fluctuating. In such a case the anterior wall of the belly is carried beyond the reach of the intestines. The intestines are tethered by the mesentery, and in the circumstances referred to the mesentery is too short to permit the intestines to reach the surface of the fluid; or the intestines may be bound down to the posterior wall of the abdomen by adhesions or a thickened mesentery, and similar symptoms result. On the other hand, the area of dulness and resonance in a case of ovarian dropsy may simulate that usually met with in ascites. The cyst may contain air either as a result of tapping or of a communication with the intestine. In these circumstances the most prominent part of the abdomen (the patient lying on her back) would be resonant; and the resonance would be altered by change of the position of the patient much as it is in ascites. In such cases the diagnosis may be impossible, though in some cases examination of the fluid drawn by tapping may supply valuable data for arriving at a correct conclusion.

In estimating the data derived from the latter source, the totality of the characters should be taken into account, and no absolute reliance should be placed upon the presence or absence of fibrin or paralbumen: for fibrin may occur in the fluids of ovarian cysts as well as in ascitic fluid, and paralbumen has been discovered in the fluid of a renal cyst. If on microscopic examination columnar epithelium be found, the fluid is ovarian. Drysdale and others believe that the so-called ovarian cell is diagnostic.

A small quantity of fluid in the peritoneum is often present with ovarian cysts. In some cases, however, there is considerable ascites, and this may greatly enhance the difficulty of diagnosis. When the ovarian tumour is small, it may under these circumstances escape detection; when large, a peculiar condition is found which may give rise to a suspicion of cancer. The diagnosis should be made from the history and after having had recourse to tapping.

Fatty or fibroid tumours of the mesentery or peritoneum occasionally attain a large size. They may simulate ovarian disease so nearly that the diagnosis may become impossible. They have been tapped under the impression that they were cystic.

Renal tumours, both cystic and solid, have been mistaken for ovarian disease, and their true nature discovered only when the abdomen has been opened with a view to ovariotomy. Renal tumours begin in the lumbar region, and grow downwards and forwards, and not upwards from the pelvis. They carry the intestines in front of them, whereas ovarian tumours as a rule push the intestines backwards; when, however, the intestines have become adherent to a tumour of the ovary, they may lie in front of the growth. When the tumour is of the left kidney the large intestine crosses it obliquely from above downwards: when of the right, it runs along the inner border of the growth. When the intestine does not contain air, it may be inflated per rectum so as to give a tympanitic note on percussion, or it may be recognised by rolling it under the finger to excite contractions of its walls. Simm recommends the introduction of elastic tubes into the intestine, to determine its relation to the tumour. In cases of renal tumours there is often a history of urinary troubles—there may be blood, pus, or albumen present in the urine.

Examination by the vagina, or with the hand introduced into the rectum, will discover whether or not the growth is connected with the organs of generation.

A moveable or floating kidney occasionally becomes considerably enlarged, and may be mistaken for an ovarian tumour; the peculiar shape of the organ is, however, preserved; the region usually occupied by the kidney is empty; the mass can be pushed up into the normal position of the kidney; and there is no connection between it and the pelvic organs.

Tumours of the liver grow from above downwards, and when they become so large as to reach the pelvis their independence of the pelvic organs can be ascertained by the hand in the rectum.

An enlarged spleen is situated on the left side, and grows downwards and towards the middle line. The elongated tumour formed by it is smooth and firm, and its right border, which runs obliquely downwards, presents the notch characteristic of the organ.

The diagnosis between ovarian cyst and pelvic hæmatocele and abscess can present difficulties only in the case of a small ovarian cyst complicated with inflammation. In such cases the history and the absolute immobility of tumours arising from exudation, sanguineous or inflammatory, will serve for distinction.

In some cases of large abdominal tumours the means enumerated will fail to effect the diagnosis, and in such cases recourse must be had to operative means, as the withdrawal of some of the fluid contents by tapping, incision of the walls of the abdomen, with a view of exploring the cavity and the nature or relations of the tumour. Even these means, however, in some very rare cases will fail to establish the diagnosis. In such cases, and indeed in all cases, the question to be decided by exploratory incision is the possibility of removal of the tumour rather than its exact origin.

PROGNOSIS.—Ovarian cysts rarely, if ever, get well spontaneously. Many never attain a large size, and are discovered on examination after death only. When they have risen from the pelvis they usually continue to grow, and ultimately prove fatal in one of the ways already mentioned. In some rare instances, however, they have ceased to grow, and have caused but little inconvenience.

TREATMENT.—The medical treatment of ovarian growths consists in attention to the general health. No medicine, as far as is at present known, has any effect upon their course. The only means that provide relief or cure are surgical—paracentesis or ovariotomy. It is beyond our province to describe these operations, and we must be satisfied with stating that ovarian cysts have been in great part deprived of their terrible character by Spencer Wells and Thomas Keith, who by their labours have rendered ovariotomy one of the most successful of capital operations.

FIBROID TUMOURS OF THE OVARY.

Fibroid tumour of the ovary is an extremely rare disease. A large uterine fibroid which in its growth has involved the ovary may readily be mistaken for an ovarian tumour, and some cases which have been reported as fibroid tumours of the ovary have in reality been tumours of the uterus. Fibroid tumours consist chiefly of fibrous tissue, and appear to be due to hypertrophy of the stroma of the ovary. Muscular fibre-cells have also been discovered in them, but in small quantities only.

Some of these growths, however, are not due to simple hypertrophy of the ovary, but are distinct nodules growing in the substance of the organ. Several such nodules may be agglomerated together and form one tumour. They vary in density, some having a hard uniformly dense structure, others containing smaller or larger loculi or cysts, while a third class possesses a loose vascular texture and present a cancerous appearance. These tumours do not usually attain a large size; they are rarely larger than a child's head. A case has been reported by Simpson, however, in which a fibroid tumour of the ovary weighed fifty-six pounds.

Like fibroid tumours of the uterus they occasionally undergo a process of calcification. They are, however, subject to far more serious changes, for instances are known in which they have become gangrenous, or have broken down and suppurated, and formed fistulous channels communicating with the vagina or elsewhere. In some cases the pedicle has been found twisted and the tumour in a congested, softened, or livid state, a condition which if left unrelieved would certainly lead to gangrene of the growth.

Pregnancy is a complication associated with the gravest danger, for during labour the tumour may be so bruised as to suppurate or become gangrenous.

The symptoms present nothing characteristic. They are such as result from the development of any innocent tumour in the pelvis.

The diagnosis is not always if ever possible with certainty, because we can never be sure that we have not to deal with a pedunculated fibroid tumour of the uterus. There are no sure means of distinguishing between the two.

If the growth is believed to be ovarian, it may be distinguished from cancer by its slow increase and its smooth surface; from cystic tumour by its hardness and absence of fluctuation.

The prognosis is unfavourable as to cure, but favourable as to the duration of life. They grow slowly and cause but little inconvenience. Like other tumours, however, they may give rise to uterine, rectal, and vesical troubles, especially when lodged in the pelvis.

The treatment should be directed to the relief of symptoms. It is only under special circumstances that recourse should be had to operative measures for their removal.

CANCER OF THE OVARY.

Cancer of the ovaries occurs but rarely as a primary disease, though it is not unfrequently seen as an affection secondary to cancer in other organs. Every form of cancer has been met with in the ovaries; but there is some difference of opinion as to whether the gelatinous or encephaloid variety of the disease be the more common. It occurs in two forms, either as nodules growing in the ovary, which may attain a large size, or as a diffuse infiltration of the whole organ. Cancer may grow also in ovarian and dermoid cysts. Cancerous tumours of the ovary vary in size from that of a walnut to that of a man's head. They generally, but not always, contract adhesions to neighbouring organs, and consequently become fixed and immoveable.

The symptoms of the disease other than those of an innocent tumour are, tenderness, pain, like the pain of cancer in other parts, rapid growth of the tumour, œdema of the pudendum, and of the lower limb on the affected side, depreciation of the general health, cancerous cachexia, and great ascites. The treatment is purely palliative.

Sarcoma is an extremely rare disease of the ovary. It appears as the spindle-celled variety, and affects both sides. It presents a cavernous structure owing to enlargement of the vessels. The only case I have seen presented a cystic character. Myo-sarcoma has been described by Spiegelberg.

It runs a rapid course, and ends fatally.

The diagnosis is difficult. Its rapid growth, tenderness, and early ascites point out its malignant character. It is said to be distinguished from cancer by the absence of metastasis; but in the only case seen by me, and already referred to, there were secondary deposits in the lung and kidney.

The treatment is palliative.

Papilloma of the ovary has been observed. It is accompanied by great ascites.

Tubercle of the ovary has no significance, except as a part of general tuberculosis.

§ VIII. DISEASES OF THE CUTANEOUS SYSTEM.

- Exanthemata.
- Squamæ.
- Papulæ.
- Vesiculæ.
- Pustulæ.
- Bullæ.
- Tubercula.
- Maculæ.
- Syphilida.
- Animal-parasite Diseases.
- Vegetable-parasite Diseases.
- Diseases Foreign to this country.

DISEASES OF THE SKIN.

By A. Balmanno Squire, M.D.

INTRODUCTORY.

In our account of the diseases of the skin we shall adhere in the main to the classification proposed at the beginning of this century by Dr. Willan. Firstly, because his classification is the one that will be the most familiar to our readers. Secondly, because it has never been supplanted in general estimation by any of the numerous systems that have since been propounded. And thirdly, because, although it is obviously imperfect (for it is an arbitrary, not a natural arrangement), we think it to be still the best medium for obtaining a clear and practical knowledge of the diseases of the skin. Cutaneous pathology has not so far advanced since Willan's time as to demand a thorough revolution in this department of medicine. But it has nevertheless made sufficient progress to require some modification of the classic system which has so long stood its ground both in this country and abroad, and whose popularity has survived so many formidable attacks.

The alterations which may be said to be *required* by the present state of science consist in the addition of three new genera, the diseases comprised in which were formerly distributed amongst the eight genera described by Willan, and were confused with other diseases from which they are known to be distinct in causation, appearance, and indications of treatment. We shall accordingly add to the items of Willan's classification (Exanthemata, Squamæ, Papulæ, Vesiculæ, Pustulæ, Bullæ, Tubercula and Maculæ), the following, viz., Syphilida, Animal-parasite eruptions, and Vegetable-parasite eruptions. And we shall make one more alteration, which we think will facilitate the attainment of a clear view of our subject, viz., the further elimination from Willan's genera of all diseases that are utterly foreign to this country, which diseases we shall group by themselves at the end of our list.

With this preface we may proceed at once to display our system of classification, and to describe the diseases included by it.

CLASSIFICATION.

Exanthemata—
Erythema, Roseola, Urticaria.

Squamæ—
Psoriasis, Pityriasis, Ichthyosis.

Papulæ—
Lichen, Strophulus, Prurigo.

Vesiculæ—
Eczema, Herpes.

Pustulæ—
Ecthyma, Impetigo.

Bullæ—
Pemphigus, Rupia.

Tubercula—
Lupus, Cheloid, Verruca, Acne, Molluscum.

Maculæ—
Nævus, Lentigo, Ephelis, Vitiligo.

Syphilida—
Vegetative, Exanthematous, Vesicular, Squamous, Papular, Pustular, Bullous, and *Tubercular Syphilides.*

Animal-parasite Diseases—
Scabies, Phthiriasis.

Vegetable-parasite Diseases—
Tinea favosa, Tinea tonsurans, Tinea decalvans, Sycosis, Chloasma.

Foreign Diseases—
Elephantiasis Græcorum, Elephantiasis Arabum, Frambœsia, Pellagra, Malum Alepporum.

SECTION I.—EXANTHEMATA.

The Rashes, or Exanthemata, are Erythema, Roseola and Urticaria.

ERYTHEMA.

Synonyms.—Inflammatory Blush. Erythème, Dartre érythémoïde, Hautröthe, Rothlausgeschwulst, Rubor, Combustio.

Definition.—Erythema (ἐρυθήμα, redness) occurs as circumscribed stains of a red colour occupying a limited portion of the skin. These patches of inflammatory redness are the seat of tingling, pricking, and smarting sensations.

Erythema may be described as of two kinds, viz., the local and the constitutional.

Under the term LOCAL ERYTHEMA are included the following varieties:—

E. simplex, E. intertrigo, E. leve, E. paratrimma, and *E. pernio.*

The varieties of CONSTITUTIONAL ERYTHEMA are:—

E. nodosum, E. papulatum, E. circinatum, and *E. fugax.*

Local Erythema.

ERYTHEMA SIMPLEX lasts usually only a few days.

Causes.—Local irritants of various kinds, such as a mustard-poultice, an irritating plaster, or the friction of the dress, producing a chafe.

In the coup-de-soleil, or sun-stroke, which, rare in this country, is so common and so fatal an accident in tropical climates, an erythema of the face and hands is commonly the first symptom (*E. solare*).

But E. simplex is most commonly the result of irritating secretions flowing over the skin. Thus it may be produced on the cheek by the discharge from the eyes in purulent ophthalmia, or in infants on the buttocks by neglect of cleanliness, or on the upper lip by the flow of acrid discharge from the nostrils during a cold, or in any situation by the discharge of pus from an ulcer or sinus.

Diagnosis.—Simple erythema of the buttocks of infants is frequently mistaken for infantile syphilis. But in the latter disease the eruption is of a ham-coloured tawny hue, and is often complicated with "mucous tubercles" around the anus and an inelastic cracked appearance of the soles and palms. It is accompanied too with snuffling at the nose and a peculiar alteration of the infant's cry.

Treatment.—The treatment of E. simplex consists in the removal of the offending cause and the use of some mildly astringent application, such as cold-cream or weak lead-lotion.

ERYTHEMA INTERTRIGO (*Intertrigo,* a chafe-gall) is only found where two opposing surfaces of the skin are in contact. For example, in the armpit, under the buttocks, under the breasts, or between the toes. It is especially liable to happen in stout people, and in them may occur in the groins or in the folds of skin about the neck. It is most common in fat infants and in stout persons, especially women of the age of from forty to fifty, and more particularly adults of the lymphatic temperament, or in children of scrofulous constitution. In these last it may be accompanied with a copious thin mattery discharge (Purifluent Erythema).

Description.—The rash begins in a rose-coloured line at the bottom of a fold of skin; it gradually extends over the whole of the crease or fold. From the resulting red surface exudes a thin, ill-smelling discharge, which is usually serous, but is occasionally purulent.

Intertrigo is accompanied with a feeling of heat and of other disagreeable sensations.

It not unfrequently lasts for some time, being often kept up by the constant rubbing that the disagreeable sensations in the part provoke. In corpulent people who are of uncleanly habits E. intertrigo may pass into the chronic state, and superficial ulcers with pale exuberant granulations may take the place of the original rash.

The *Treatment* of this variety of Erythema consists in separating the contiguous surfaces by keeping a piece of scorched rag between them, or in dusting them over with some soft impalpable powder, such as violet-powder or lycopodium-dust, which absorbs the irritating moisture; or the secretion may be constantly kept away by frequent washing of the surfaces, or may be checked by the use of astringent lotions. Painting the surface with tincture of iodine often has a very excellent effect.

Erythema leve is produced when the skin stretched over an œdematous part inflames. This variety of Erythema occurs for the most part in the legs, but when the patient is confined to bed it may occur in other (dependent) situations.

Description.—It appears as smooth, shining red spots, accompanied with increased swelling of the subjacent areolar tissue. This variety of Erythema derives its importance from the tendency of the patches to terminate in gangrene of the affected skin and of the subjacent areolar tissue. When this is about to take place the patches assume a purplish hue, and serous blebs are formed over them.

The *Treatment* of E. leve consists in removing the cause (œdema) by diuretics, sudorifics, or aperients, by position or by acupuncture, and in using stimulating applications, such as camphorated spirit or tincture of iodine, to the erythematous patches.

Erythema paratrimma (or the *Bedsore*), happens in the course of debilitating diseases that confine the patient long to bed, such as typhus, or typhoid fever, or paralysis of the lower half of the body.

The common position of the bedsore is over the sacrum, but it is occasionally situated over either of the trochanters or of the iliac crests.

Description.—It appears at first as a diffused dusky redness of a limited portion of the skin, attended with a sensation of pricking; sometimes serous blebs are developed on the reddened surface; soon the affected skin becomes blackened, and a more or less extensive slough forms, which sometimes extends very deeply.

The *Treatment* consists in the removal of pressure from the affected skin; in preventing the flow of irritating secretions over it; in a scrupulous attention to cleanliness, and in the local use of stimulating applications.

If a slough has formed, its separation should be favoured by the application of yeast poultices. After the separation of the slough, the ulcer should be dressed with some moderately stimulating ointment.

Erythema pernio (or the *Chilblain*) is a disease almost peculiar to children and young persons, affecting especially those who are of lymphatic temperament.

Chilblains are most common in children under ten years of age. The usual situation of chilblains is on the backs of the toes and of the fingers, although after puberty they are found also on the nose. They happen generally after a thaw, or as a consequence of warming the hands or feet by the fire when they are cold and damp; in some individuals they appear regularly every winter, disappearing in the spring.

Description.—Chilblains appear as shining, red, swollen patches which are the seat of severe itching and tingling, and are extremely sensitive to even the slightest pressure. These after continuing for a few days may gradually subside, leaving only a slight scurfiness of the affected skin.

But if they persist for many days, the reddened patches assume a bluish tint and become surmounted by the watery blebs or "blains" from which the complaint derives its name.

These blebs become ruptured and ulceration takes place, and so a foul-looking wound with ragged edges and a slough-like floor is produced in the situation of the erythematous patch. This ulcer (commonly known as the broken chilblain) is sometimes pretty deep, and is always slow to heal.

In the *Treatment* of chilblains, measures must be adopted to increase the activity of the general circulation by a generous and stimulating diet, active exercise, frictions of the skin with hair-gloves, &c.; and at the same time activity of the circulation in the affected part should be specially promoted by the use of stimulating applications, such as soap-liniment or camphor-cerate. If the chilblain be "broken" resin-ointment will be a suitable dressing; poultices are to be avoided if possible.

Rheumatic Erythema.

Under this heading are included all except one of the varieties previously enumerated as coming under the category of constitutional Erythema.

ERYTHEMA NODOSUM is a disease almost peculiar to young persons, more particularly those of lymphatic temperament. It attacks especially young girls, in whom it is frequently associated with amenorrhœa and chlorosis. It is the most common of the three varieties of rheumatic Erythema. It appears to be sometimes occasioned by fatigue, exposure to cold, and insufficient nourishment.

The appearance of the eruption is usually preceded for a few days by slight feverishness and rheumatic pains in the joints. These pains often continue with the eruption and may even outlast it. They are sometimes very severe.

Description.—The eruption affects almost invariably the legs, attacking especially the fore-surface of the leg, but it occurs sometimes on the backs of the fore-arms and on the thighs.

It presents the appearance of red, elevated patches of an oval form. The long diameter of the patch is generally parallel with the axis of the limb. The patches vary from an inch to three inches in diameter. They are palpable swellings, but their margin is ill-defined. At first they are hard and tender to the touch, and are of a rose-red colour, but in a few days they soften and change to a violet colour; in a few days more this fades into a yellow tint, and the elevation disappears.

Each patch lasts on the average a week, but the disease is generally by a succession of patches prolonged for two or three weeks.

Treatment.—The treatment of E. nodosum should be commenced by a dose of aperient medicine. The patient should be put to bed, or at all events directed to lie on a sofa most of the day. Quinine conjoined—if there be much anæmia—with iron should be taken regularly in tolerably large doses, and if there be any indication of a scrofulous taint, the addition of cod-liver oil will be of service.

ERYTHEMA PAPULATUM differs from E. nodosum only in the much smaller size of its patches, which rarely exceed the size of a split-pea, and in the situations occupied by it (the face, neck, breast, and arms). In every other respect its history is that of E. nodosum, and it demands the same treatment. It is of less common occurrence than E. nodosum.

Sometimes the spots are larger, from the size of a sixpence to that of a shilling, and proportionately elevated; they are then apt to occur also on the legs (E. tuberculatum). Such spots form a connecting link between this variety and the preceding one.

ERYTHEMA CIRCINATUM appears at first as small round patches, slightly raised above the level of the surrounding skin. These gradually spread at their circumference, and at the same time fade at their centre, and so assume the form of red embossed rings, the outer margin of the ring being abrupt and well-defined, while the inner edge of the ring is gradually shaded off. The area enclosed by the ring is of a faded yellow colour. The breadth of the ring itself varies from a quarter to half an inch; generally several rings are developed one after another, some of them may attain a diameter of several inches. After they have spread to a certain extent, portions of the rings are apt to fade, and the remaining portions, uniting with similar fragments of other rings, form with them variously-curved lines.

This variety of Erythema is confined generally to the trunk. It lasts about a week. It is not a common eruption. It sometimes occurs in the course of an attack of acute or sub-acute rheumatism. It requires the same treatment as E. nodosum.

Dyspeptic Erythema.

ERYTHEMA FUGAX is characterised by the sudden appearance of large red patches usually on the face, but sometimes on other parts, namely, on the arms and upper part of the trunk. After remaining out for a few hours they as suddenly disappear. In a day or two they reappear either in the same place or in a new one.

This eruption forms the connecting link between Erythema and Urticaria, and is by some writers included under the latter heading. It is generally brought on by some error of diet.

The *Treatment* of it consists in remedying whatever disorder of the digestive organs may be the cause of its appearance.

ROSEOLA.[1]

Synonyms.—Rose-rash; False Measles; Roseole; Fièvre rouge; Eruption rosacée; Fausse rougeole; Rötheln; Rotherhund.

Definition.—Roseola, Roseus, rosy, is a rash which presents the appearance of a diffused mottling of the skin spread over a considerable extent of surface; the mottling being produced by small rose-coloured patches, which, running into one another, enclose islets of unaffected skin.

Causation.—Roseola is more common in the summer (R. æstiva) than in the winter, and seems to depend often on the disorder of the stomach and bowels so common at that season.

It is of commoner occurrence in infants (R. infantilis), than in children or grown-up persons, being provoked often by the disorder of the alimentary canal that very frequently attends the first dentition.

In infants the patches are smaller and more closely arranged than in the adult.

In adults it is sometimes excited by the drinking freely of cold water after violent exertion.

Roseola is sometimes occasioned by vaccination, and sometimes occurs in the course of smallpox, and other exanthemata.

Description.—The eruption of Roseola is preceded for a day or two by slight febrile disturbance, which subsides as the rash becomes developed, and there is commonly some dryness and redness of the fauces.

The network of inflammatory redness presented by roseola may commence on the chest, the belly, or the upper limbs, and may remain limited to either of those regions, or may spread all over the body.

The duration of the rash is very brief. After persisting for three or four days, it fades away, leaving sometimes a slight scurfiness of the affected skin. Its colour is deepest on the second day.

When Roseola is produced by vaccination (R. vaccinia), it appears at the same time that the "red areola" forms around the vaccine vesicles, and spreads from the "areola" over a considerable extent of surface, sometimes even covering the whole of the body. Its appearance increases the (usually) slight febrile disturbance that accompanies the formation of the "areola."

When Roseola occurs in the course of smallpox (R. variolosa), the appearance of the proper smallpox-eruption is delayed, the roseolous rash appearing at the time that the pustular smallpox-eruption should, and for two or three days taking the place of it. This occurrence is more common in inoculated than in natural smallpox.

The variolous Roseola, like the ordinary eruption of smallpox, spreads from the face and chest to the extremities. It is sometimes so abundant that the affected surface looks almost uniformly red, and the case is then very apt to be mistaken for scarlatina. But the appearance of the pustular smallpox-eruption very soon corrects an error of this kind.

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[1] See also vol. i. Art. Roseola

Diagnosis.—Roseola is so apt to be mistaken for measles as to have received the name of *false measles.* In either disease there is a diffused rash all over the body, interspersed with intervals of natural skin.

The rash of measles is, however, of a mulberry rather than of a rose colour. Its reddened patches exhibit a crescentic shape. They appear first on the forehead, whereas Roseola may commence on other parts of the body, and when it begins on the face, spreads from the neighbourhood of the nose.

There is (in measles) more severe disturbance of the system. The feverishness precedes the eruption for three days (instead of only one). The eyes are swollen and watery in measles; and, lastly, measles is a very highly contagious disease, which Roseola is not.

"Syphilitic roseola" may be distinguished from Roseola properly so-called by the dusky and sombre hue of the rash, by the absence of itching, by the longer continuance of the rash, and by the more serious character of the throat-lesion when the throat is affected.

Prognosis.—Roseola is generally a brief and trivial disorder, occasioning but little inconvenience, and terminating within a week after its commencement. Sometimes, but very rarely, it assumes a chronic form, appearing and disappearing at uncertain intervals.

Treatment.—Roseola is a disease which, on account of its trivial character and its usually brief duration, does not require very active interference. Indeed it derives its chief importance from its liability to be confounded with other more serious complaints. A restricted and simple diet, gentle saline aperients, and a few warm baths, are in most cases all that is required.

If there be much acidity of the stomach, magnesia is indicated.

In infantile Roseola, if the gums be hot and tender, they should be lanced.

When the disease is chronic, the internal administration of dilute nitro-hydrochloric acid is of service, but sometimes a change of air and a course of sea-bathing are necessary.

URTICARIA.

Synonyms.—Nettle-rash; Nettle-fever; Fièvre ortiée; Porcelaine; Nesselausschlag; Nesselfieber; Cnidosis; Uredo; Porcellana; Aspretudo; Febris urticata; Essera; Lora.

Definition.—Urticaria (*Urtica urens,* the stinging-nettle) is an eruption which resembles the effect produced on the skin by the contact of the stinging-nettle, and which is accompanied by the burning, tingling sensation that the sting of the nettle occasions. It is characterised by evanescent cutaneous elevations called "wheals" or "pomphi," which may be described as circumscribed swellings of the skin, more or less hard, sometimes whiter, sometimes redder than the healthy skin. They are sudden, both to appear and to disappear.

Causes (Predisposing).—A special predisposition or idiosyncracy; infancy, childhood, and adolescence; fineness and delicacy of the skin; the female sex; a gouty habit.

The exciting causes of Urticaria are: the spring season; sudden exposure to cold during warm weather; high living and intemperance; over-exercise; strong mental excitement; dentition; morbid affections of the uterus and the urinary organs; derangements of the digestive organs. The ingestion of certain aliments, amongst which may be enumerated lobsters, mussels, cray-fish, crabs, shrimps, smoked or salted fish, mushrooms, cucumbers, strawberries, raspberries, honey, bitter almonds, sweet almonds, oatmeal, pickles, caviare, pork, eggs, beer, coffee, liqueurs, seltzer-water. The ingestion of certain drugs, such as valerian, copaiba, capsicum, iodide of potassium and the turpentine group of drugs. The contact of certain irritants with the skin: for example, of the urtica urens, the loasa, and several other stinging plants; of some caterpillars; and of certain molluscs, such as the "Portuguese men-of-war;" the bites of gnats, mosquitoes, fleas, bugs, &c.; the irritation set up by the pediculus corporis.

But it must be remarked that the majority of the exciting causes above enumerated can operate only on persons of special susceptibility, and that people in whom Urticaria is readily excited by some of these causes are quite insensible to the operation of others. For instance, certain persons who can never eat eggs without experiencing a consequent attack of Urticaria may nevertheless be able to partake of strawberries with impunity, and *vice versâ*.

Description.—Urticaria may be either acute or chronic. In the acute form, which is the commoner, the duration of the eruption varies from a few hours to a few days; whereas chronic urticaria may last for several months, or even several years.

In either case the eruption is essentially the same. The wheals or pomphi may vary in breadth from that of a millet-seed to that of a crown-piece. When of the smallest size they present the appearance of papules rather than of pomphi, and appear more prominent than the broader patches ("Lichen urticatus"). The larger wheals often present a distinct depression at their centre. Their outline, which is always well defined, may be either regularly rounded or irregularly sinuous, so that they resemble the islands on a geographical map. Sometimes they occur in long bands (U. gyrata), and look like the marks of a whip. The surface of the wheals (excepting those of the smallest size) is flattened. The wheals are sometimes scattered thinly over the affected surface, but are sometimes so numerous and so thickly set as to become confluent (U. conferta). Their colour is generally a dull white, but often a rosy or a crimson red. In either case they are commonly surrounded by a rosy areola, so that when the pomphi are at all thickly set, the areolæ of neighbouring pomphi blend with one another. When the pomphi, as commonly happens, are of a dull white colour, the surrounding rosy areola, which commences abruptly at the margin of the wheal, gives it a very conspicuous appearance. In regions where the subcutaneous cellular tissue is loose, the eruption may be complicated by considerable subcutaneous swelling. Thus the face and the scrotum, when attacked by an eruption of Urticaria, occasionally become so much swollen as to present a very alarming appearance.

Although the course of the eruption may extend over a considerable space of time, the duration of an individual pomphus is always very brief, varying from a few minutes to, at the most, a few hours. The sudden appearance of the pomphi, their transitory existence, and their abrupt subsidence, constitute one of the most striking peculiarities of the disease.

Varieties.—The rash may be either acute or chronic.

Acute Urticaria is of two kinds. In the one (U. febrilis), the appearance of the rash is preceded for two or three days by feverishness; in the other (U. ab ingestis), the eruption occurs within an hour or two after the ingestion of some offending substance.

In the former kind, commonly known as the Nettle-fever, the disease begins with some fever; the patient complains of headache, nausea, and pain in the epigastrium; he feels faint and languid, and his tongue is furred. After these symptoms have continued for two or three days, they are terminated by the appearance of the rash, the development of which is attended with much tingling and excessive itching. These sensations provoke the patient to rub or scratch himself, and thereby greatly to increase the eruption. The attack lasts generally a week or two, with frequent fluctuations in the condition of the rash. The wheals are usually more developed in the evening than at other times of the day. Whenever, during the course of the attack, the rash recedes, the symptoms (already referred to) which ushered it in are apt to recur.

The Urticaria ab ingestis comes on much more suddenly than an attack of Nettle-fever. It lasts a much shorter time; but, while it lasts, is a far more severe illness. It often occasions great alarm by giving rise to the supposition that the person attacked by it has been poisoned, and so indeed he may be truly said to have been, although his actual danger is by no means in proportion to the severity of his symptoms. An hour or two after the offending substance has been swallowed, pain is felt at the epigastrium. This is accompanied with nausea and faintness; there is much heat, and tingling, and intolerable itching of the skin; and soon the eruption makes its appearance. It is very confluent, and is attended with considerable subcutaneous swelling. It attacks chiefly the face, neck, and upper half of the body, but sometimes extends over the whole surface. Occasionally the mucous membrane of the mouth and pharynx is much swollen, and the patient sometimes suffers from dyspnœa, so as to seem almost on the verge of suffocation. After a few hours' duration the violence of the attack subsides, and in a day or two the patient is well again. If, as often happens, vomiting or diarrhœa occur during the course of the attack, great amelioration is at once produced. The most alarming symptom in an attack of this kind is the occurrence of extreme prostration.

Chronic Urticaria is unattended with general symptoms. The eruption is not so violent as in the acute variety. Nor are the wheals quite so evanescent as those of acute Urticaria. However, in the chronic, as in the acute kind, the wheals have individually but a brief duration; they are constantly disappearing and giving place to others, and so seem to be always shifting their position. As in the acute (Nettle-fever) kind, the rash is constantly varying in extent; it appears and disappears irregularly; in some cases it is "regularly" intermittent; it may last indefinitely.

Sometimes the wheals, instead of being flat and but slightly raised, project more boldly, and are of a hemispherical shape, attaining the size of a nut, or even that of a walnut (U. tuberosa).

A large proportion of the cases of chronic Urticaria that occur in hospital practice, are dependent on the presence of the pediculus corporis.

Urticaria more commonly affects the shoulders, the loins, the thighs, the face, and the fore-arms.

Diagnosis.—Urticaria can scarcely be confounded with any other species of eruption. The prominent hard wheals, their evanescent character, and the sensations that accompany them, are in most cases sufficient to distinguish this disease from all others. Urticaria tuberosa may, however, present some resemblance to Erythema nodosum. But the course of the latter is more acute, its progress more continuous, and the development and decline of its individual patches take place more gradually.

Prognosis.—Although always a very annoying and often an obstinate complaint, Urticaria is scarcely ever attended with any danger. Even in severe cases of "U. ab ingestis," when the disease certainly puts on a most alarming appearance, it has scarcely ever been known to lead to a fatal issue; and in those rare cases where it has seemed to be the cause of death, the result is to be attributed rather to the internal disorder produced by the ingested substance than to the effect of the rash.

Treatment.—In "Nettle-fever" rest should be enjoined, the diet should be restricted, and mild aperients and cooling salines should be prescribed. If there be any over-acidity of the stomach, magnesia should be given; and if there be much fever it should be combated by small doses of tartar-emetic. When the eruption appears to result from the ingestion of some noxious article of diet, an emetic should be given as soon as possible, and it should be followed by some efficient purge. If there be much depression, a dose or two of chloric æther will be serviceable. The patient should be put to bed, and restricted for some little time to bland mucilaginous drinks. In either variety of the acute form, it is better to dispense with local applications.

When the disease is chronic, it sometimes happens that there is some offending article in the patient's diet, and that he requires only to abstain from this particular kind of food in order to be quit of his eruption. When this is so, the offending cause varies in different cases, and is not always easy of discovery. Willan proposes that each article of food or drink that the patient has been in the habit of taking, should be successively omitted for a short time from his diet, so that in this way the noxious substance may be detected. In some persons pork will turn out to be the cause of the rash, in some milk, in others eggs, and so forth. In some chronic cases, the internal administration of the mineral acids will be of service, and with this may be advantageously conjoined the use of mineral acid baths. But when the eruption is associated with habitual over-acidity of the stomach, the alkaline carbonates, administered both internally and externally, are indicated.

When the complaint assumes an intermittent type, quinine should be given in tolerably large doses. If the patient be of a plethoric habit, he should be restricted to a bread and milk diet for some time.

In some obstinate cases arsenic is a valuable remedy; vapour baths and the vapour douche are often useful in severe cases of long standing.

Urticarious patches, the result of local irritants, must be dealt with according to the nature of the irritation that has produced them; thus the leaves of the "dock" are the proverbial antidote to the sting of the nettle. The stings of insects (gnats, ants, wasps, &c.) derive their pungency from the injection of a small quantity of formic acid into the puncture made in the skin, and the application of a dilute solution of ammonia or potash relieves the pain of them almost directly.

SECTION II.—SQUAMÆ.

The Squamous, or Scaly eruptions, are, Psoriasis, Pityriasis, and Ichthyosis.

PSORIASIS.

Synonyms.—Dartre lichénöide; Herpes furfurans; Dartre sèche; Dry scaly tetter; Lepra vulgaris; Dartre squammeuse sèche; Schuppenflechte.

Definition.—Psoriasis (*ψώρα*, a scurvy) may be defined as a disease of the skin, in which large thick white nacreous scales cover and adhere closely to a thickened, creased, tawny-red patch of skin.

Causes.—Psoriasis is a constitutional disease of the skin.

The predisposing causes are a robust and vigorous constitution, adolescence, or adult age, and in some cases hereditary predisposition.

The exciting causes are mental emotions or anxiety; the abuse of alcoholic liquors; the seasons of spring and autumn; local injury or irritation.

It is not a contagious disease.

General Description.—Psoriasis commences in small white spots of the size of a pin's head. If one of these small scaly incrustations be detached by the finger-nail, it will be found to be pretty firmly adherent to the surface and to conceal a minute reddened, slightly elevated patch of skin. These spots gradually increase in diameter and form discs, which vary in size from that of a pea to that of a half-crown. At the same time the squamous exudation becomes thicker and more opaque, and exhibits a mother-of-pearl-like or even silvery lustre. At a little distance the skin exhibits the appearance of having been splashed with mortar. If a separated crust of about the size of a shilling be broken across it may be seen to be of about the thickness of cardboard, and to be made up of thin dry imbricated scales arranged in strata one above the other. The skin disclosed by the removal of the crust is slightly elevated. On pinching it up between the forefinger and thumb it may be felt to be thicker and less supple than the unaffected portions of skin. It is of a tawny-red colour. On making pressure on it for a moment with the forefinger, the redness temporarily disappears, leaving however a light yellow stain. Its surface is somewhat creased.

As the spots continue to increase in size, those which are near to one another may coalesce so as to form irregular patches of some extent; these are more frequently found on the upper part of the front of the leg or on the back of the fore-arm.

Not uncommonly a spot as it spreads at its circumference heals at its centre, so as to form an annular patch of the disease. These rings as they increase in diameter may undergo various changes in form; thus part of the circle may heal, while the rest of it spreads; or, the ring remaining unbroken, some parts of it may extend their distance from the centre more rapidly than others; or two neighbouring rings may unite. By such accidents are produced figures of a semicircular, an oval, or a horse-shoe form; or the disease may describe figures of 8, or be so fantastically arranged as to give the skin much of the appearance of a geographical map. In some rare instances, by the fortuitous coalescence of several incomplete circles, a serpentine band of some length is produced.

Psoriasis usually makes its appearance first at the elbows and knees, and when it has become spread more extensively it still remains worst at those places. It appears next on the back and afterwards on the chest and belly. It is not uncommon on the hairy scalp, and on the palms of the hands and the soles of the feet; on the face, where it is comparatively rare, it is seen more commonly on the upper part of the forehead and on the eyebrows. Psoriasis is usually attended with more or less itching. It is not unfrequently found associated with eczema.

Duration.—Psoriasis follows an extremely chronic course, and its tendency is to recur again and again after recovery. It may last a few weeks, a few months, a few years, or even the greater part of a lifetime.

Termination.—When the disease is about to disappear the patches gradually get smaller, the scaly incrustations thinner, and the skin beneath them loses its undue thickness and tawny-red colour, and regains its elasticity. As a rule, the disease disappears first from the chest and abdomen if it be already present in those situations. Next it deserts the back, and lastly the limbs; traces of it still continuing linger for some time about the elbows and knees after every vestige of it elsewhere has disappeared.

Diagnosis.—Psoriasis is recognised by the dryness, the thickness, the opacity, the sheen, and the imbricated arrangement of its scales; by the thickened, inelastic, dry, wrinkled condition and tawny-red colou of the skin underneath them, and by the predilection of the diseas for the neighbourhood of the knees and elbows; it may however b mistaken for Pityriasis, but in this disease there is much less thickening and furrowing of the skin, and the scales are much scantier thinner, and finer than those of Psoriasis.

Eczema, which in its squamous stage may be thought to bear som resemblance to Psoriasis, may be distinguished by the moist appearance and thinness of its scales, and the readiness with which the may be detached from the skin. Circumscribed or annular patches o Lichen are to be distinguished by the paucity of the scales and thei want of opacity and lustre, as well as by the more rugged appearanc of the affected skin, from similar patches of Psoriasis.

In squamous Syphilide—the so-called syphilitic Psoriasis—th patches do not attain the size that is commonly reached by those o simple Psoriasis. The scales too are smaller, thinner, scantier, an are confined to the middle of the patch. The tawny-red skin i smooth and shining, in place of being rough and creased; the skin affection is usually accompanied by other symptoms of secondar syphilis. It should be noticed that a tawny hue of the inflamed skin which is so commonly relied on as the means of recognising syphilitic eruption, is common to syphilitic and non-syphiliti Psoriasis.

Prognosis.—This is always favourable when the disease occurs in mild form and is submitted to proper treatment. Its liability to reappear after it has been cured must however be kept in mind. Whe the patient is advanced in life and the disease is of long standing an extensively spread, it may resist treatment for several years.

Varieties.—When the spots of Psoriasis barely exceed the size of pin's-head the disease is called *P. punctata;* when they have attaine the size of a split-pea or a threepenny-piece, it is called *P. guttata* and when the rounded patches range in diameter from that of sixpenny-piece to that of half-a-crown, the term *P. nummularis* i employed. Where the disease presents itself in extensive irregula patches, the epithet *diffusa* is applied to it. When it occurs in rings or exhibits the traces of an annular arrangement, it is distinguished by the term *P. circinata*. If its mode of disposition should chance to produce a serpentine band, it is said to be an example of *P. gyrata*. *P. inveterata* is a term applied to the disease as it is sometimes met with in persons who have had numerous and severe attacks of it; when the greater part of the surface is covered with a thick, scaly incrustation, and the thickened inelastic skin is chapped or even deeply fissured opposite the joints, *P. palmaris et plantaris*. Psoriasis when it affects the palms and soles is characterised by larger, thicker, and less lustrous scales than it produces in other situations, and by deep painful fissures in the skin, which exude a serous or sanguineous fluid.

P. unguium.—When the nails are affected they become thickened, opaque, sometimes greyish in colour, deeply grooved often, and not unfrequently pitted in a peculiar manner; they may become more or less laminated, and in some cases the nail gets replaced by a scaly incrustation. Psoriasis when it affects the face (*P. faciei*), which however is not a common situation for it, assumes much of the appearance of inflamed circumscribed Pityriasis, the scales being small and thin, and only scantily concealing the tawny-red patches of skin to which they adhere. Psoriasis of the hairy scalp (*P. capitis*), is peculiar in this respect, that while Pityriasis, an apparently much less serious disease, is frequently accompanied with considerable thinning of the hair, Psoriasis, the crusts of which are often of some thickness in this region, entails no loss of hair.

Therapeutics.—If the eruption be acute, that is to say if it be both recent and extensive, and the patches much inflamed, refrigerant medicines and emollient applications should be employed, and at the same time the diet should be restricted. When the inflammatory appearance of the disease has by such means been reduced, recourse should be had to the liquor arsenicalis (Ph. Br.) in doses of from ♏ij to ♏v thrice daily. In cases where the inflammatory excitement of the patches has a tendency to reappear, it will be advisble to make the addition of a small dose of the potassio-tartrate of antimony, or to replace the liquor arsenicalis by Donovan's solution. Free phosphorus in doses of $\frac{1}{10}$ grain (t.d.s.) dissolved in oil is sometimes a most efficacious remedy. In some cases, however, these remedies are inadmissible on account of their giving rise to gastro-intestinal inflammation, and the same objection applies to another remedy of almost equal repute—the tincture of cantharides, which has the additional disadvantage of causing irritation of the genito-urinary tract. Under such circumstances it is necessary to rely chiefly on external applications. Ointments of the green iodide or the ammonio-chloride of mercury, of the former in the proportion of from ten to thirty grains to the ounce, of the latter in the ratio of from a scruple to a drachm to the ounce, are certainly of great efficacy in the treatment of Psoriasis; but when, as is often the case, the disease occupies a large extent of surface, their use is very apt to occasion salivation. The various tarry preparations have a great and well-merited reputation as topical applications in Psoriasis. The unguentum picis liquidæ (Ph. Br.) dilute with twice the quantity, or with an equal proportion of lard, o applied *per se*, or ointments of the huile de Cade, or of the empyreumatic oil of the Betula alba in the same proportions, or ointments o creosote or of resin, may be employed; they are open, however, to on therapeutical objection, and that is, that in some persons they are ap to give rise to an erysipelatous inflammation of the skin, which has anything but a favourable influence on the progress of the disease. Thi is to be guarded against by the prudent use of a weak ointment t commence with, but the pungent smell of these substances, and th fact that they stain and corrode the linen of the patient, are furthe drawbacks. In the matter of smell the oil of the Betula alba is perhaps the least objectionable, and creosote has the advantage of not staining the linen.

Chrysophanic acid (derived from Goa-powder) in the form of ointment (ʒss to ℥j) has recently been used with considerable success, but it is apt to produce conjunctivitis unless care be taken in its use; waterproof (vulcanized india-rubber) underclothing in some cases of itself effects a cure. Prolonged immersion, namely, for several hours daily, in a simple tepid bath at the temperature of 92° Fahr. will in some instances cure the disease.

PITYRIASIS.

Synonyms.—Porrigo furfurans; Dartre furfuracée volante; Teigne amiantacée; Dartre farineuse; Dandriff; Kleienflechte; Herpes furfurans.

Definition.—Pityriasis (πίτυρον, bran) is a disease in which loosely-adherent minute, thin, opaque, white scales cover a harsh surface of skin.

Causes.—Pityriasis is a constitutional disease which is often transmitted hereditarily. It occurs on the scalp, more commonly in females than in males, and in persons who wear their hair long than in those who keep it cut short. Children from the age of six to that of puberty are especially liable to it. It affects the dark rather than the fair.

The immediate causes to which it has been traced are a previous eczema of the same portion of skin, over-feeding, great fatigue, and mental anxiety.

General Description.—The commencement of the disease is a dry harsh condition of the skin which loses its natural softness and suppleness. Soon very dry, minute, thin, opaque white scales make their appearance on it; they are readily detached even by the slightest friction, or even fall spontaneously, but are renewed as fast as they fall off. Sometimes the skin is reddened as well as harsh, and may even be slightly thickened and creased so as to approach the condition already described as proper to Psoriasis. There is usually more or less itching of the affected skin, but unless the surface involved be extensive and considerably inflamed there is no constitutional disturbance.

The *varieties* of Pityriasis are P. alba, P. fusca, P. rubra, and P. pilaris.

Pityriasis alba attacks the scalp and (less commonly) the hairy parts of the face, and is characterised usually by an *abundant* furfuraceous desquamation; although there is generally no redness there is always considerable itching of the affected skin. The scales are readily detached in great numbers by scratching or rubbing the part; they are thin, dry, opaque, and white. This affection does not necessarily entail baldness, nor does the hair in any instance fall spontaneously, but in many cases it becomes extremely dry and brittle, so that if at all long it comes off in great quantities when combed. The baldness so produced is only temporary; when the disease ceases the hair grows again as thickly as before.

P. fusca occurs on the face and neck in the form of rounded patches, which vary in size from that of a fourpenny-piece to that of a crown. The skin, which is a little thickened and slightly creased, is of a faint tawny-red colour, and is sprinkled over with minute, white, loosely-adherent farinaceous scales, which are neither so large nor so opaque as those of P. alba. This variety occasions scarcely any itching, but when irritated the patches are liable to much smarting and burning. These sensations are generally provoked by sitting before the fire, by facing a cold wind, or by washing the face with soap and water.

P. rubra is a less common variety than either of the preceding. It is sometimes accompanied with slight febrile disturbance. The scales are larger and more adherent than in P. fusca, and the skin is much reddened. The disease is situated generally on the chest, but is sometimes extensively spread over the upper part of the body and the upper limbs. It is accompanied by itching and tingling.

P. pilaris is the rarest variety of all; it affects only the orifices of the hair-follicles, leaving the intervening skin untouched. It may invade every region except the scalp. The orifices of the hair-follicles, more particularly of those situated on the outer surfaces of the limbs, become thickened and prominent, and the root of each hair gets surrounded by a small, hard, conical elevation, composed of minute adherent scales; the skin feels dry, harsh, and rough, and presents the appearance commonly known under the name of goose-skin. There is rarely either redness, itching, or tingling of the diseased surface. Pityriasis pilaris is an extremely chronic and obstinate affection. It is generally associated with P. rubra of the chest and arms.

Duration.—Pityriasis generally follows a chronic course, lasting for several months, or even many years. In children, however, its duration is often limited to a few weeks.

Diagnosis.—Pityriasis is to be distinguished from *Psoriasis* by the scales in the former disease being small, thin, bran-like, and but loosely adherent to the skin; while in the latter they are large, thick, adherent, and nacreous. The considerable infiltration and thickening of the cutis in Psoriasis, and the predilection of that disease for the knees and elbows, are further means of diagnosis. A *squamou eczema* may in many respects resemble Pityriasis, but the mois character of the former, as well as the greater size, the yellowness and the semi-translucency of its scales, will readily distinguish it *Herpes circinatus* is a disease with which P. fusca is very apt to be confounded; but the more perfectly circular shape of the patches of Herpes, and their well-defined edges, which are more elevated and scaly than their central portions, or a microscopical examination of the scales, will generally suffice for its recognition. *Ichthyosis* may easily be known from Pityriasis by its history alone, since it is always a congenital disease.

The *Prognosis*, except in the case of P. pilaris, is always favourable; but all varieties of the disease are apt to return after recovery.

Therapeutics.—In Pityriasis of the scalp or of the hairy parts of the face, the first thing to be done is to have the hair cut short. If the whiskers or beard be affected, they should be trimmed close with scissors, not shaved with a razor. In any case where the scalp is affected, and hair-cutting objected to, all combing of the hair, and any mode of dressing it which pulls it much about, should be interdicted.

The Pityriasis of the scalp which occurs in infants, and in which there is considerable admixture of sebaceous matter with the scales, merely requires that the head should be kept well cleansed, brushed often and lightly with a soft brush, which, acting as a gentle stimulus to the skin, soon restores it to its normal condition.

The local applications most in use for Pityriasis are the vapour-douche, alkaline-baths, sulphuretted-baths, alkaline-lotions (sodæ subcarb. gr. v. to ℥j.), a solution of soft-soap, lotions of nitric-acid (♏xx to ℥j.), of acetic-acid, of hepar sulphuris; ointments of alum, of acetate of lead, of sulphate of zinc, of ammonio-chloride of mercury, of tar, of nitric-acid (♏xx to ℥j.).

The internal remedies are the various sulphuretted mineral waters, the liquor arsenicalis, bitters—hops, centaury, chiretta, gentian—sulphur.

The diet should be of the simplest kind.

ICHTHYOSIS.

Synonyms.—Ichthyose; Fish-skin disease; Die Fischschuppenkrankheit; Lepra ichthyosis; Der Fischschuppenaussatz; Maladie écailleuse; Serpentine; Pargamine.

Definition.—Ichthyosis (ἰχθυς, a fish) is a peculiar congenital condition of the skin, in which the epidermis, in place of being smooth, unctuous, soft, and elastic, is dry, harsh, rough, and unyielding. This condition is a permanent one.

Causes.—The disease is congenital, and is in many cases hereditary, sometimes missing a generation. It has been observed by Cazenave to be commoner in certain districts of France than in others, and by Biett that females are less liable to it than males.

General Description.—Ichthyosis occurs as a peculiar scaly condition of the skin, in which there is neither redness of the cutis nor any sensation either of heat, itching, or tingling. The scales are pretty firmly adherent to the surface on which they rest, and are not readily detached. If pulled off, their separation does not occasion any pain, and the surface disclosed is neither rough, as in Psoriasis, nor moist, as in Eczema. In many cases the formation of scales is complicated with an excessive development of pigment. The disease is sometimes general, sometimes local. When general, the palms of the hands, the soles of the feet, the arm-pits, the groins, and the eyelids are always left unaffected. When partial, it affects especially the limbs, more particularly the neighbourhood of the knees and elbows.

On different parts of the same individual it sometimes presents widely different appearances. Whatever may be its intensity, or for however long a period it may have existed, it exercises no perceptible influence on the general health. Although it never entirely ceases, it is sometimes greatly modified under the influence of the seasons; it is generally less in summer than in winter. This is doubtless due to the larger quantity of liquid secreted by the skin in warm weather.

The *varieties* are I. serpentina, I. nitida, and I. cornea.

I. serpentina is so called from the skin in this variety resembling in appearance the belly of a lizard or of a snake. The epidermis is dry and glossy, and is divided into little angular pieces by a number of fissures, which decussate with one another.

In *I. nitida* the skin is much like that of a fish. The scales have a mother-of-pearl-like glitter. They are larger than in the preceding variety, and thicker; they have an imbricated arrangement. Sometimes the scales, instead of being pearly-white, are of a dark-grey colour.

In *I. cornea* the epidermis is much hardened, greatly thickened, and deeply fissured, so as to form a series of closely-packed, horny, mobile excrescences, which are usually of a dark olive-brown colour. These are sometimes so long and so hard, that by passing the finger-tip rapidly over the surface, a dry rattling sound may be produced. Persons affected with this variety of Ichthyosis sometimes exhibit themselves as a public show, under the name of porcupine-men.

Diagnosis.—The only two diseases with which Ichthyosis is likely to be confounded are *Psoriasis* and *Pityriasis.* From the former, however, it differs in being usually extended over a large surface—sometimes over nearly the whole of the body; while Psoriasis occurs in the form of patches, which are separated from one another by intervals of sound skin. Again, when a portion of skin is affected with Ichthyosis, there is no definite limit to the disease; it fades by insensible gradations into sound skin. The scurf of Pityriasis in its fineness, but more especially in the slightness of its adherence to the skin, differs widely enough from the scales of Ichthyosis. Again, the absence of all inflammation of the skin and of the itching and other unpleasant sensations that attend it serve to distinguish Ichthyosis from either of the two other squamous diseases.

Therapeutics.—Although the treatment of Ichthyosis cannot, at least in the present state of our knowledge, be radically curative, still much may be done in the way of palliative treatment. For the serpentine and nacreous varieties the most efficacious remedies are in the first instance baths, lotions, or ointments of the alkaline carbonates; and when by such means the skin has been temporarily restored to a healthy condition, this may often be maintained by keeping the surface slightly greased with almond- or olive-oil, which should be washed off at least twice a week. Cod-liver-oil has, on theoretical grounds, been recommended in place of sweet-oil, but no real advantage is to be obtained by the substitution, and the nauseous smell of the fish-oil is much against it. Vapour-baths, vapour-douches, or warm-water douches may take the place of alkaline applications, and glycerine may be used instead of oil. The administration of cod-liver-oil or of arsenic internally, or of tar both internally and externally, has been advocated, but the efficacy of these remedies is very doubtful. In the horny variety the treatment should be commenced by painting limited portions of the surface in succession with blistering-fluid. When by this means the horny excrescences have been removed, the skin should be treated as in the other varieties.

SECTION III.—PAPULÆ.

The Papular eruptions are, Lichen, Strophulus, and Prurigo.

LICHEN.

Synonyms.—Dry itch; Dartre furfuracée volante, Poussée; Gale sèche; Schwindflechte; Papulæ; Herpes siccus; Tinea volatica; Papulæ siccæ; Exormia lichen; Scabies sicca; Licheniasis adultorum; Scabies agria.

Definition.—Lichen (λειχὴν, a tree-moss[1]) is an eruption of numerous minute red papules clustered together, attended with much itching, and succeeded by a harsh, wrinkled, thickened condition of the skin.

Causes.—Lichen is met with at all ages, but affects preferably adults. It is commoner with females than males, and in spring and autumn than in summer or winter. Persons of nervous temperament are especially liable to it. It is sometimes hereditary. Certain occupations may be looked upon as predisposing causes, viz., those which subject any part of the skin to the constant action of irritants—*e.g.* that of a pastrycook, a grocer, a bricklayer, a metal-worker, or a dyer; and those which entail frequent exposure to the glare of a furnace—*e.g.* that of a cook, a smith, a metal-founder, or a stoker. Overfeeding, drunkenness, too rich a diet, a sudden chill, watchfulness, and depressing mental emotions, such as anxiety or grief, are also to be ranked among the causes of Lichen.

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[1] This is the derivation usually given. More correctly however the primitive meaning of λειχὴν is a skin-disease, and it is derived from λείχω to lick (as animals lick a sore), so that the skin-disease was not viewed as a moss on the skin, but rather the tree-moss was regarded as a skin-disease of the tree.

Description.—The disease at its commencement consists of an eruption of minute red papules clustered together on an inflamed portion of skin. On opening one of these papules with the point of a needle it is found to be solid, containing neither serum nor pus. On passing the finger over the affected skin, it is felt to be dry and rough. After a short time the tops of the papules either become covered with white, branny scales; or, getting excoriated by repeated scratching, they exude minute drops of serum or blood, which dry up speedily into small, hard, grey, or black crusts. At the same time the skin becomes thickened, its natural linear markings or wrinkles get deepened, and it loses its elasticity; so that if the affected part be in a position where it is constantly liable to be stretched, it "parts" opposite one or more of these furrows, which thus become converted into cracks or even deep fissures. This is a common result when the disease occupies the back of the wrist. The increased thickness of the diseased skin may be readily felt if a portion of it be pinched up between the finger and thumb. If the direction of the little furrows be examined, it will be found that they are arranged so as to cut the surface of the skin into minute fusiform islets.

The *varieties* of Lichen are L. simplex, L. circumscriptus, L. agrius, and L. inveteratus.

The term *simplex* is applied to the disease when it presents the characters above described in a comparatively mild degree, is diffused over a surface of some little extent, and has no definite outline; *circumscriptus*, when it occurs in rounded patches (of the average size of a five-shilling-piece), with well-defined outlines; *agrius*, when the eruption is acute, is ushered in with febrile symptoms, and partakes in some degree of the character of Eczema or of Impetigo; that is to say, there is an admixture of vesicles or of pustules with the papules; *inveteratus*, when the disease has become very chronic and severe. This variety is characterised by considerable thickening of the skin and unusual depth of the furrows on its surface. In some cases of L. inveteratus the scales that are formed on the diseased surface are so copious and so thick, as to give the disease much of the appearance of Psoriasis. Other terms have been applied to Lichen: *lividus*, when occurring in cachectic individuals the papules are livid; *gyratus*, when the disease forms sinuous bands; *tropicus*, which distinguishes a variety resembling L. simplex that is common in tropical climates.

What has been termed *L. urticatus* is a variety of Urticaria. "*L. pilaris*," again, is a form of Pityriasis.

The itching and burning sensations that accompany Lichen are usually severe, but they are especially so in cases of L. inveteratus. They always become aggravated towards the evening. The disease occurs most commonly in persons of nervous or sanguine temperament; it is confined to no special period of life; it affects chiefly the backs of the hands and wrists, the back and sides of the neck, the front of the thighs, and the back of the trunk; it is least frequently seen on the hairy scalp. It is a constitutional disease; follows a chronic far more commonly than an acute course, and is apt to recur again and again after apparently perfect recovery. It is not contagious.

Diagnosis.—L. simplex may be mistaken for Prurigo, Scabies, or Strophulus; L. agrius for Eczema; and L. circumscriptus for Herpes circinatus, Pityriasis, or Psoriasis: but

In *Prurigo* the papules are large, flat, isolated, and most of them are covered with a small black crust.

In *Scabies* the situation of the eruption, its multiform character, and the presence of the acarian furrows are distinctive signs.

Strophulus affects only young children. It is always an ephemeral eruption. The papules are larger than in Lichen, and are usually scattered loosely over the surface. When grouped, the papules in each group are few in number, and the patches are small. There is no induration or furrowing of the skin in Strophulus.

Eczema may be distinguished by its appearing at first in the shape of vesicles instead of papules; subsequently by the even surface, superficial excoriations, abundant plastic serous secretion, broad, moist, yellow, flaky incrustations, and thinner state of the skin, which contrast with the roughened, dry, papulated surface, the small, dry, grey crusts and thickened cutis of Lichen.

Herpes circinatus may be known by its shape being more circular than that of L. circumscriptus, its scales thinner and softer, and its centre less rugous; moreover, it spreads with much greater rapidity. In doubtful cases a microscopical examination of the scales may be had recourse to.

Pityriasis, when it occurs in reddened, circumscribed patches, with slight thickening and wrinkling of the skin, may resemble L. circumscriptus, but there is generally but little if any itching in this form of Pityriasis. The induration and thickening of the skin is much les marked, and the scales are much more readily detached than in Lichen

In *Psoriasis* the skin is considerably thickened and wrinkled, bu not to the extent seen in L. circumscriptus. The scales, on the othe hand, are much thicker and more copious, and exhibit a special silver lustre; the disease moreover affects especially the elbows and knees.

Prognosis.—Lichen is not in any way dangerous to life; but it obstinate character, its tendency to relapse after recovery, and th severe itching that accompanies it, render it a serious affection.

Treatment.—In an ordinary acute attack of Lichen but little treatment is required. A moderate and unstimulating diet, the avoidanc of alcoholic liquids, and the wearing of soft underclothing, are sometimes all that is necessary. If feverishness attend the complain saline laxatives and refrigerant drinks may be given; and if th affected skin be very irritable, emollient tepid baths, containing bran, starch, or gelatine, or lotions of infusion of linseed, of solution of acetate of ammonia, of lime-water, of diluted vinegar, of mist. amygd. amar., or of *weak* hydrocyanic-acid solution may be used. In a severe attack of L. agrius it may be necessary to enjoin a very restricted diet, to apply leeches in the neighbourhood of the inflamed surface, and to employ starch- or bread-poultices. In a less severe attack, excluding the surface from the air by means of the blandest applications, such as sweet almond-oil, cold-cream, glycerine; or by dusting it over with lycopodium may at the outset suffice for local treatment. If, when the inflammation has decreased, the disease exhibits a tendency to assume a chronic condition, much benefit is often to be derived from the use of alkaline-baths, which may contain an ounce of the carbonate of potash or of soda to the gallon of water. In some cases baths of sulphuret of potassium may be substituted with advantage. These measures should be conjoined with the internal administration of the fixed alkalies or their carbonates.

In obstinate cases of Lichen, where the skin has become much thickened and is desquamating, lotions of liq. potassæ, ʒss.–℥ss. to ℥j. aq. (the stronger solutions requiring great caution in their use), sulphur vapour-baths, the vapour-douche, ointments of calomel, camphor, tannin, the green iodide of mercury, the bichloride of mercury or tar, are proper applications. Internally small doses of Donovan's solution or of some one of the sulphurous mineral waters should be given. Cases of chronic Lichen that are attended with debility, as in L. lividus, are greatly benefited by such tonics as the nitro-muriatic acid or the triple citrate of quinine, iron, and strychnia. Cauterization with nitrate of silver is useful in some cases of persistent L. circumscriptus, and in similar cases the use of chrysophanic acid ointment (gr. x–ʒij. to the oz.) has proved serviceable. In some cases the wearing of waterproof underclothing over the patch or patches is of considerable benefit. Free phosphorus taken internally (dissolved in oil) in doses of gr. $\frac{1}{50}$ t.d.s. proves in some instances a very efficacious remedy.

STROPHULUS.

Synonyms.—Red Gum; Tooth Rash; Feu des dents; Lichen Strophulus; Exanthema Strophulus; Rothe.

Definition.—Strophulus (στρέφω, to torment) is an acute eruption, accompanied with itching, which appears in the form of slightly acuminated papules of the size of a pin's-head or that of a millet-seed, distinct from one another, and either whiter or redder than the surrounding skin.

Causes.—The disease is peculiar to infancy and childhood; it is of most frequent occurrence in the first two years of life. This is doubtless owing to the vascularity of the skin of infants, and the excitability in them of the nervous centres concerned in reflex action.

immediate causes of Strophulus are errors of diet in the mother or nurse while suckling, or the more direct administration of unsuit- food, over-clothing, rough flannel next the skin, uncleanliness, nement to close and heated apartments, and, most of all, the constitutional disturbance produced by the eruption of the first set of teeth.

Description.—The *varieties* of Strophulus are S. intertinctus, S. confertus, S. volaticus, S. candidus, and S. albidus.

S. intertinctus is characterised by papulæ of a vivid red colour, inkled scantily over the surface, distinct from one another, often ermingled with erythematous spots or patches. In *S. confertus* the papulæ are smaller and are less red than those of the preceding variety, but are more numerous, and are often clustered together. *S. volaticus* appears as small red rounded patches, each of which is ade up of a few papules, closely grouped together. These patches st only for a day or two, and disappear at one spot to reappear at another. In *S. candidus* the papules are large and distinct; they have a smooth and shining surface, and are lighter coloured than the surrounding skin. They are not, however, surrounded at their base by any inflammatory areola.

The above varieties of Strophulus are attended with slight feverishs, and, when dependent either on visceral derangement or dentition, h the symptoms peculiar to those causes respectively. The average uration of the eruption is about a week, but it occasionally lasts for as long as three weeks. When it disappears it is succeeded by a slight ephemeral furfuraceous desquamation.

Strophulus affects chiefly the exposed parts of the surface, the face, fore-arms, and backs of the hands, but occurs commonly on other parts of the body.

S. albidus was classed under the genus Strophulus, on account of its possessing what were thought to be the essential characters of that affection, namely, that it consists of small discrete papules, and is an infantile disease. However, in more important details it differs widely from the other species of Strophulus. Thus it is a chronic and indolent affection, produces no itching, and is in no way connected either with gastro-intestinal disorder, or with the process of teething. Again, the substance of the papules instead of being made up of infiltrated fibro-cellular tissue, consists of a collection of inspissated sebaceous follicles. They occur principally on the face and appear as small opaque, white, elevated specks, which may or may not be surrounded by a slight erythematous ring. They are readily distinguished from S. candidus by the characters already mentioned, by their much smaller size, and by their *opaque* white appearance. They often coincide with the other varieties of Strophulus, although, as above stated, they are due to a separate cause. They result from the activity of the sebaceous function of the skin in the infant.

Diagnosis.—The diseases for which Strophulus is most likely to be mistaken are Scabies, Lichen, and Urticaria, but in infants the eruption of *Scabies* on the hands and feet is usually vesicular, and the acarian furrows are readily found; the eruption generally appears first on the nates; the history of contagion is readily made out, and the nurse is almost invariably affected. In *Lichen* the papules are small and clustered together, and in young children the eruption is generally widely spread, so that a large continuous surface becomes harsh, rough, and inflamed. In *Urticaria* the papules are larger and flatter, and are mingled with still larger irregularly-shaped pomphi. They are very ephemeral, disappearing suddenly at one place and reappearing as suddenly at another. This latter character, it is true, is shared also by S. volaticus, which approaches nearly in nature to Urticaria; but the diagnosis between the two is of but little practical moment, since they are both benefited by the same measures, and are of similar origin and nature.

Prognosis.—Strophulus is a trivial affection, which rarely lasts longer than a week or two, and is not prone to recur.

Treatment.—Tepid glycerine-baths, or sponging with tepid milk-and-water, and powdering the skin with starch or lycopodium, are generally all that is necessary. If the child is suckling, its nurse should take some saline refrigerant drink. If the gums are swollen and inflamed at any part they should be lanced. When the eruption is connected, as it often is, with acidity of the stomach, this should be corrected by some one of the alkaline carbonates—carbonate of magnesia, of soda, or of lime being selected, according to the state of the bowels, aided, if need be, by some carminative. The room should be kept cool, and the infant's clothing should be light and soft.

PRURIGO.

Synonyms.—Pruritus; Cresmos; Prurit; Psore papuleuse; Scabies papuliformis.

Definition.—Prurigo (Prurigo an itch) is a disease characterised by an eruption of comparatively large, but only slightly elevated flat papules of the same colour as the surrounding skin, distinct from one another, covered at their tops by small black crusts, and accompanied by more or less intense itching. It is in some instances contagious.

Causes (of general Prurigo)—The pediculus corporis, poverty, dissipation, neglect of cleanliness, drunkenness, old age, exhausting chronic diseases. [The acarus scabiei also produces a species of general Prurigo. This is treated of under the head of Scabies q.v.] (Of Prurigo pubis)—The pediculus pubis. (Of Prurigo podicis and scroti)—A sedentary life, a too rich and stimulating diet, habitual constipation, ascarides in the rectum, a chronically-congested condition of the rectum, internal piles, urinary calculi, old age. (Of Prurigo pudendi muliebris)—Leucorrhœa, uterine disease, the "change of life," ascarides in the rectum, a feeble constitution.

Description.—Prurigo may be widely diffused over a large extent of surface, or it may be confined to a very limited area. When of the *ffused kind* it commences by itching of some region which at once vokes the patient to scratch. Soon an eruption of small, scattered, papules arises; the tops of these being caught by the patient's nails, are soon scratched off; a slight extravasation of blood ensues, which, coagulating, forms the small black crusts which cap the papules. The itching, which is increased towards the evening, but is always easily supportable, becomes gradually extended over a greater area. In this condition the disease is called *P. mitis.*

In a severer form (*P. formicans*) the irritation is more intense, and is of a different character. The itching is accompanied by pricking and burning sensations, and a feeling as if a multitude of ants were crawling over the skin. It gradually increases in severity and extent, so as to be almost insupportable. The patient is irresistibly impelled to scratch, and seeks relief by tearing the skin deeply with his nails. The irritation is always most severe in the evening and the early part of the night, which is generally far advanced before the sufferer gets any rest. He may even be altogether deprived of sleep for several nights in succession. It is augmented too by the ingestion of a full meal, or of any spirituous drink, or by the warmth of a fire, and I have noticed that it is always much worse in summer than in winter. In this variety the papules are broader and flatter, and are surmounted by thicker and blacker crusts than in P. mitis. They are moreover intermingled with deep, linear excoriations, sealed by streaks of black coagulum, which are the marks left by the patient's nails.

In a variety yet more severe (*P. senilis*), which is found chiefly in old persons, the itching gets gradually more and more distressing, the papular eruption becomes more extensively spread, and the marks of the patient's nails deeper. The skin becomes at some points blackened and rugous, at others dark-coloured. The papular eruption gets intermingled in some places with ecthymatous pustules, in others with urticarious wheals and fugacious erythematous patches. The worry of mind and the loss of sleep entailed by this frightful disorder seldom fail to impair in some measure the general health. A patient who has suffered long from the complaint generally wears a haggard and anxious face. In some instances the prolonged and constant torment has led the sufferer to commit suicide.

General Prurigo is seen at all ages. In children and young persons it assumes usually the form of P. mitis, in middle-aged adults that of P. formicans, and in the aged the condition known as P. senilis. As regards its situation, the regions chiefly affected by it are—according to my own researches—either the back of the neck and of the shoulders, together with the upper portion of the front of the chest, or the small of the back, together with the nates and anterior and outer surfaces of the thighs. It may be very severe in either of these two situations, without being very marked in the other. I have seen it occupying all parts of the body, except the hairy scalp, the face, the armpits, the fingers, the palms of the hands, and the soles of the feet. The disease is very chronic, lasting for months or years, and is often extremely intractable. In many cases it appears to be caused and maintained solely by the presence of the body-louse (Pediculus corporis).

A form of Prurigo (*P. pubis*) which holds a middle place between the diffused and the circumscribed kinds is that occasioned by the pediculus pubis or crab-louse. This commences over the pubic region and gradually extends itself upwards over the abdomen and downwards over the thighs. This affection is usually confined to the hairy parts of the anterior surface of the trunk and thighs, and rarely extends downwards below the middle of the legs or upwards above the axillæ. The itching occasioned by it is usually pretty severe, but the eruption consists only of scattered papules smaller in size than those of the varieties already described, and surmounted by redder crusts and of small papular urticarious pomphi. There are none of the deep linear excoriations which form so prominent a feature in P. formicans and P. senilis. It, in fact, resembles papular scabies more than any other disease.

The more important of the circumscribed *varieties* of Prurigo are P. podicis, P. scroti, and P. pudendi muliebris.

P. podicis, or itching of the skin around the anus, is often a most tormenting and intractable affection. The irritation, which is very intense, is constant, but is augmented in the evening and early part of the night. In some cases there is considerable itching of the margin of the anus itself, as well as of the skin in its vicinity. The furious scratching that this complaint provokes generally produces a sero-mucous discharge from the anus, which is attended with some relief. When the affection has lasted for some time the skin becomes considerably thickened as well as harsh and inflamed, so as to resemble in some measure chronic Lichen, and the itching gets complicated with severe burning and pricking sensations.

In *P. scroti* there is intense irritation of the scrotum, generally of its posterior surface, accompanied often at first by a sero-sudorific discharge, and later by induration, reddening, dryness, and harshness of the affected skin. This variety often coincides with, and is sometimes merely an extension of P. podicis.

In *P. pudendi muliebris* the parts affected are the labia majora and minora, and sometimes also the lower part of the vagina. The irritation is if possible more severe and more intractable than in either of the two preceding varieties. The impulse to scratch the itching part is so urgent and so frequent as to drive the patient from society, and in some instances to give rise to nymphomania. The appearance of the parts in some cases remains unaltered; in others there is some fulness and redness of the mucous surface, and occasionally minute abrasions, which exude a serous fluid, may be seen.

Diagnosis.—In general Prurigo the large, flat, slightly elevated, isolated papules, of the same colour as the surrounding skin, covered with small black crusts of dried blood, are sufficiently distinctive to prevent confusion with any other disease.

In *Lichen* the papules are very small and acuminated, grouped closely together, of a red colour, and covered with little grey crusts or with a furfuraceous desquamation.

In *Scabies* the different general arrangement of the eruption, its multiform character, and the presence of the acarian furrows, will serve to distinguish it from Prurigo, but the most ready way of distinguishing practically between the two (one that the author believes he is the first to point out) is that while in Scabies the fingers are always more or less affected, in Prurigo, although the backs of the hands may occasionally be attacked, the fingers *never* are.

Strophulus is a disease peculiar to children; the papules are either much redder or much whiter than the surrounding skin; they are never surmounted by black crusts.

Prognosis.—Under proper treatment Prurigo sometimes disappears in two or three weeks, but it is often extremely chronic, lasting for months or even years. Although not in itself directly fatal, it may lead indirectly to a fatal issue. The constant mental and bodily distress occasioned by it is apt to break down the health, produce considerable emaciation, and render the subject of it more liable to be cut off by other diseases; and the despairing condition of mind sometimes induced by the long-continued, tormenting sensations, may even lead to suicide.

Treatment.—This will vary greatly according to the cause. In *general Prurigo* an over-excitable condition of the skin may be calmed by lotions of alum, acetate of lead, corrosive sublimate, chloroform, æther, or hydrocyanic-acid; by ointments of belladonna or opium; by dusting the surface with oxide of zinc; by baths of lime-water, carbonate of soda, dilute nitric-acid, vinegar, alum, or of corrosive sublimate; by the use of tar-ointment; by the employment of soft-soap, or by prolonged daily immersion in tepid-baths; and by the internal administration of opium, belladonna, stramonium, or aconite in suitable doses. When the disease is associated with pediculi, a cerate made with the fixed oil of stavesacre-seeds, as first proposed by the author, or an ointment of the flowers of the Pyrethrum album, of precipitated sulphur, of the sulphate or sulphuret of mercury, of iodide of potassium, or of bichloride of mercury, the use of sulphuretted-water or vapour-baths, or of cinnabar vapour-baths, or the Harrogate or Moffat waters are the most efficient remedies. Scrupulous attention should be paid to cleanliness, and the patient's underclothes frequently changed. When the general health is much impaired, attention should be paid to the diet, which should be nourishing but unstimulating; bitter tonics, steel, cod-liver-oil, &c., should be administered; the patient should use tepid sea-salt baths, and take regular exercise in the open air. There are various empirical modes of treatment which have found favour with some. These are the internal administration of nitrate of silver, of arsenic, of sarsaparilla, of the decoction of dulcamara, of the alkaline carbonates, of the iodide of potassium, &c. The author's experience of these, however, is not such as to give him faith in their efficacy.

In *P. pubis* a lotion of hyd. bichlor. gr. ij, ad ℥ aq., or an ointment of hyd. am. chlor., or dusting the surface lightly with calomel, at once cures the disease. The first effect of either of these applications is that the moribund pediculi cause more irritation even than before, but in the course of two or three hours the irritation ceases altogether.

In *P. podicis* it will sometimes be advisable to apply leeches round the margin of the anus; cold hip-baths, cold lotions, anodyne lotions, are generally of service. An ointment of acetate of morphia, made with cold-cream, or a suppository of tannin and morphia made with cacao-butter, will often afford great relief. Citrine-ointment is also a useful application. Internally the most efficient remedies are occasional small doses of calomel, the nitro-muriatic acid, podophyllin, sal ammoniac, the extract of taraxacum. A mixture of mag. sulph. with acid sulph. dil. in infusion of roses taken thrice daily is sometimes of value. It is of great importance in this affection to regulate the diet, both as regards its quantity, which should be restricted, and its quality, which should be unstimulating.

In *P. scroti* the same plan of treatment should be pursued as in P. podicis.

In *P. pudendi muliebris* any uterine disease or irregularity should be inquired into and treated. The most efficient local applications are a dilute solution of corrosive sublimate, a lotion of calomel and limewater, or a solution of chlorate of potash. Internally various anodynes and nervine tonics may be given, such as small doses of aconite, hyoscyamus, assafœtida, or oxide of zinc.

As before stated, severe itching of the perinæum is sometimes dependent on the presence of ascarides in the rectum. In the female these parasites not unfrequently crawl from the anus to the vulva, and give rise to distressing irritation in both situations. In such cases the treatment of the affection is confined to the removal of the cause.

SECTION IV.—VESICULÆ.

The Vesicular eruptions are, Eczema and Herpes.

ECZEMA.

Synonyms.—Humid Tetter; Moist scaly Tetter; Running Scall; Dartre vive; Dartre squammeuse humide; Poussée; Schwestblättern; Hitzblätterchen; Eczesis; Herpes squamosus madidans; Ruborescum vesiculis et pruritu.

Definition.—Eczema (ἐκζέω, to boil out oı over) is an eruption characterised at its commencement by the development of numerous minute agglomerated transparent vesicles, or by a multitude of small red fissures in the epidermis; afterwards by a superficially-excoriated, moist reddened surface, exuding more or less abundantly a clear plastic serum, which concretes into buff-coloured flaky crusts, and at its termination by a scaly desquamation of the epidermis.

Causes (Predisposing).—Eczema occurs at all ages, but is commoner with the young than with the aged; young children and infants are especially liable to it. It becomes more prevalent at the spring, and again towards the end of the autumn, than at other times of the year. Certain avocations which entail much exposure to the rays of the sun, or to the heat of an oven, or of a furnace, or which necessitate much handling of gritty or irritating substances, predispose to Eczema. Thus it is of common occurrence amongst agricultural labourers, metal-founders, blacksmiths, pastrycooks, bakers, colour-grinders, dyers, metal-workers, and grocers. But independently of these causes there is evidently in certain individuals a strong *constitutional* predisposition to the disease which in many instances is hereditary.

The exciting causes of Eczema are the process of dentition, excesses of all kinds, the ingestion of shell-fish or highly-spiced dishes, strong mental emotions whether of an exciting or a depressing kind, want of sleep, excessive fatigue, prolonged exposure to the direct rays of the sun in hot weather, the contact of irritating plasters, of rancid oil, of strong alkali, of mercurial ointment, or of irritating powders, the irritation set up by the presence of the acarus scabiei, or the pediculus corporis.

Description.—Eczema may follow either an acute or a chronic course.

Acute Eczema in its mildest form (E. simplex) occurs without previous constitutional disturbance, in the form of slightly-reddened patches, on which are speedily developed numerous minute glistening vesicles containing clear serum. These vesicles have but a short existence. In the course of a day or two, either they subside in consequence of the fluid within them becoming reabsorbed, or they become ruptured and allow it to exude. In the one case the vesicles are replaced by minute furfuraceous scales, in the other by small thin crusts. Whether scales or crusts be formed they soon separate, leaving the skin perfectly sound. The eruption is attended with slight itching and tingling. Its whole course occupies about a week or ten days.

In a severer form (*E. rubrum*) the phenomena of inflammation are more strongly marked. The eruption is preceded by considerable febrile disturbance. The inflamed patches are not only vividly red, but are often considerably swollen, and on examination by touch feel hot and tense. As in the milder variety the reddened skin is thickly sprinkled with minute transparent vesicles; these may either be succeeded by desquamation, the skin remaining red for some days after their disappearance, or may give rise to a copious exudation of plastic serum, which concretes into thin, moist, cheesy flakes, which are repeatedly cast off and renewed, the skin continuing for several days red, moist, and excoriated. After a time however it becomes drier and gradually loses its redness, while the flaky crusts are replaced by a dry furfuraceous desquamation, and at the expiration of two or three weeks the diseased surface resumes its healthy appearance.

Chronic Eczema.—This consists either of a prolonged series of subacute attacks or of a permanently-diseased condition of the skin. It presents itself under a variety of shapes. In some cases the most prominent phenomenon is an abundant secretion of clear, thin, serous discharge, the diseased skin looking red, swollen, and sodden.

In others the liquid secreted is less abundant but more glutinous, exuding from a number of minute superficial excoriations, or else from a number of little red decussating fissures in the epidermis.

Sometimes the secretion is scantier still, and at the same time of a thicker consistence, so that instead of issuing from the affected skin as a colourless tenacious fluid, it concretes almost as soon as it is formed into thin, yellow, soft, somewhat moist exfoliations. These are not very adherent to the skin beneath in consequence of its comparatively moist condition, but whenever they get detached they are speedily replaced.

In other cases there is but little tendency to a moist secretion; the reddened skin is covered with yellowish semi-transparent flakes, which are pretty firmly adherent to it except at their margins, which are detached, white, and opaque.

In others again the principal character of the disease is a thinned, smooth, tense, and shining condition of the skin, which is sprinkled over with flakes so thin and transparent as to be barely perceptible.

Besides the varieties of form above described, Eczema presents in special situations certain special peculiarities.

Thus on the hand (*E. manuum*) acute Eczema appears in the form of large vesicles which vary in size from that of a millet-seed to that of a split-pea; they usually occur in groups, and are sometimes confluent. Their appearance is attended with much heat and tingling; usually no *itching* is complained of until they have begun to disappear. If the eruption be copious the hands will be red, swollen, and aching. Usually the vesicles do not rupture, but after remaining for some days shrink up and are replaced by a dry desquamation. However, where the epidermis is comparatively thin—for instance, on the lateral surfaces of the fingers or of the hand, or on the backs of the finger-webs—they may be succeeded by the yellow, moist exfoliations that are ordinarily produced by Eczema.

When chronic Eczema occupies a position where the skin is frequently stretched and relaxed by the movements of the part covered by it—*e.g.* the palm of the hand, the knuckle or flexure of a finger, the angles of the mouth, the margin of the anus, &c. — the red fissures, in place of being (as before described in the general account of Eczema) numerous and merely epidermic, are few and extend deeply into the cutis.

When the eruption is situated near any one of the orifices of the body, it is apt to extend inwards along the mucous membrane of the passage; and conversely certain inflamed conditions of the mucous passages are apt to extend in the form of an eczematous eruption ove the skin which is continuous with them. Thus the author has see an Eczema of the skin around the mouth result from an ulceration o the buccal mucous membrane, caused by the contact of a carious toot Very often Eczema of the skin covering the eyelids will extend ove the palpebral conjunctiva in the form of granular ophthalmia ; and o the other hand granular ophthalmia will extend in the form of Eczem over the integument of the eyelids and cheek. Eczema of the pinn of the ear, again, frequently extends up the meatus externus, and ma produce either a form of otorrhœa or thickening of the membran tympani, and so some degree of deafness. Eczema of the external ea moreover leads to considerable induration and swelling of the pinn which becomes tender, rigid, and more or less altered in shape.

Chronic Eczema of the leg depends in many cases on a varicos state of the veins of the limb, but whether arising from this cause o not, it is apt to leave behind it a more or less persistent brown discoloration of the skin.

When it attacks the hairy parts of the body (scalp, axillæ, or pubes the viscid serous discharge of Eczema, which in these situations usually pretty abundant, either keeps the hair wet, sticky, and entangled as happens when the secretion is profuse and comparatively thin ; or less copious and of a thicker consistence it forms, with the entangle hair, dry, firmly-adherent crusts.

Of Eczema of the hairy scalp there are, according to the author researches, two distinct kinds. The one, which may be distinguishe as the constitutional variety, affects chiefly the anterior half of th hairy scalp, and is generally confined to that portion of it that cover the expansions of the frontal and temporal bones. This species ofte coincides with E. aurium, and not unfrequently extends over the forehead. It occasions in many instances considerable thinning of th hair. It is sometimes an obstinate affection, and is apt to recur afte recovery. The other variety, which may be termed occipital Eczem occupies chiefly the posterior half of the scalp, and is generally confined to the part covering the occipital bone. This kind as a rule not found associated with Eczema of any other part. It is almos peculiar to children between the ages of two and sixteen year whereas the constitutional variety is common at all periods after th age of three months. The occipital variety is invariably associate with and depends on the presence of the pediculus capitis; in th constitutional kind, unless it be associated with the other affection the pediculus is as constantly absent. The occipital variety moreove is always readily to be got rid of, and does not commonly recur ; does not produce thinning of the hair.

The Eczematous eruption caused by the *Acarus Scabiei* will be found described under the head of "Scabies."

Eczema is by far the commonest disease of the skin. It is ofte found associated with some other cutaneous affection, the eruption in such cases partaking of the characters of either disease. Lichen, Impetigo, Pityriasis, and Psoriasis are the diseases with which it is most frequently conjoined. It follows usually a chronic course, and is very apt to return after recovery. It is attended generally with a sensation of burning and itching, and causes occasionally severe smarting, or even aching pains. It is in most cases a constitutional complaint, but sometimes arises solely from parasitic irritation.

Diagnosis.—In considering the diagnosis of Eczema, the frequency with which it coincides with other affections of the skin must be remembered; those with which it is most commonly associated have been already mentioned. The diseases for which it is most likely to be mistaken are Lichen, Psoriasis, a variety of Erythema, Herpes, Impetigo, and (foliaceous) Pemphigus. In certain situations too it may be confounded with Pityriasis, Erysipelas, bullous Pemphigus, or Scabies. But—

In *Lichen* the quantity of fluid secreted is very small, and the crusts formed by it are minute, dry, and of a greyish colour; the skin too is harsh, dry, and thickened, whereas in Eczema it is smooth, moist, and thinned. In *Psoriasis* the incrustations are dry, white, laminated, and nacreous, and the inflamed skin covered by them is somewhat elevated, dry, creased, and of a tawny-red colour. The eruption of Psoriasis, moreover, is usually most developed in the neighbourhood of the knees, elbows, and loins. *Erythema intertrigo* may be distinguished by its position (in some fold of the skin), by the thinness of the discharge, and by the ready disappearance of the eruption under treatment. *Herpes* may be diagnosed from Eczema by the larger size of its vesicles, their occurrence in patches of about a dozen or more clustered on circumscribed inflamed areolæ, and their leaving adherent, dry crusts instead of loose, moist, exfoliations behind them. In *Impetigo* the crusts are much thicker than in Eczema; their surface is nodulated; on detaching them a suppurating surface is disclosed; in its early stage the disease is pustular. In *foliaceous Pemphigus* the eruption is more extensive than it ever is in Eczema, the exfoliations are much larger, the liquid secreted by the inflamed skin is less viscid, and there are generally a few bullæ present to aid the diagnosis.

Eczema of the scalp under certain conditions so closely resembles *Pityriasis* as to be scarcely distinguishable from it, but in the latter affection the scales are always drier and more opaque than in the former. E. rubrum affecting the face may be mistaken for *Erysipelas*, but the latter affection occupies, at its first appearance, only a very limited area—generally the bridge of the nose; the swelling ceases suddenly at the margin of the inflamed patch, and in place of a multitude of small vesicles there are one or two irregularly-shaped blebs. In acute Eczema of the hands, by the union of several vesicles blebs may be produced sufficiently large to simulate the *bullæ of Pemphigus;* but the blebs so formed are often loculated; clusters of vesicles are to be seen in their neighbourhood; they disappear in the course of a few days, and are not succeeded by others: whereas Pemphigus is almost always a chronic disease. Eczema of the hands is sometimes merely a symptom of Scabies. When this is the case, the eruption is confin chiefly to the interdigital webs and the lateral surfaces of the fir phalanges, whereas, according to the author's observations, Eczema constitutional origin attacks the dorsal and ventral rather than t lateral aspect of the fingers; again, the former never affects the la phalanges, the latter often does so. The history of contagion, whe this can be made out, and the presence of the acarian furrows, will further means of identifying Scabies.

Prognosis.—Eczema in no way tends to a fatal issue, but it ru usually an extremely chronic course, and is often a very intractab and distressing affection; it is prone to recur repeatedly after apparentl complete recovery, and at each return to become less and less amenabl to treatment.

Treatment.—If Eczema be developed under any external influenc *e.g.* the irritation produced by constantly handling sugar or lime, th removal of the cause will often alone suffice. In such cases, if th patient be a grocer or builder, it is sometimes necessary that he shoul change his occupation.

If the eruption be recent and attended with inflammatory symptoms after a brisk purge, refrigerants and demulcents, such as cream o tartar, mindererus spirit, lemonade, sarsaparilla, or couch-grass te should be given, at the same time that emollient and sedative applications are employed. For instance, if the eruption be general, tepid-baths containing bran or gelatine; or if it be limited to a small area, poultices of ground-rice or potato-starch, made with an infusion o henbane or lettuce. The use of an evaporating lotion composed of a drachm of Goulard's extract to a pint of distilled water is often an efficient remedy; this lotion being used in the daytime and replaced at night by an ointment containing six drops of the liquor plumbi in an ounce of "vaseline."

If the disease be chronic and the patient of lymphatic temperament, moderate doses (℥ij t.d.s.) of cod-liver-oil should be taken; in long-standing cases of this kind considerable benefit is to be derived from small doses of sulphur or of the hepar sulphuris. In the majority o instances the liquor arsenicalis will be of service. Free phosphorus in doses of gr. $\frac{1}{30}$ (dissolved in oil) t.d.s. is also a remedy of unquestionable value. When there is considerable watery discharge the bowel should be kept loose by a saline aperient. If the patient's health b feeble he should take steel and bitters, and his diet should be nourishing, but in any case it should be unstimulating, and all condiments an fermented liquors should be interdicted.

Locally various remedies may be made use of with a view to thei exercising either a stimulant, an alterative, an anodyne, an astringent or an emollient effect on the skin. And in a disease which varies s much in its characters as Eczema the success of any plan of treatmen will depend greatly on the judicious selection of the local applicatio and the appropriate adjustment of its strength.

Ointments of calomel, of the nitrate or the subiodide of mercur of vermilion, of oxide of zinc, of tannin, of camphor, of calamine, of cyanide of potassium; or lotions of borax, potash, bichloride of mercury, acetate of lead, sulphate of iron, nitrate of silver, and the huile de Cade, are the most important topical remedies. The unguentum diachyli of von Hebra (made by softening diachylon plaster with an equal quantity of linseed-oil) is a favourite and useful remedy; so also is the glycerole of the subacetate of lead (*i.e.* the liquor plumbi made with glycerine in place of water) diluted with from one to seven parts of pure glycerine.

HERPES.

Synonyms.—Tetter; Dartre; Zittermahl flechte; Ignis sacer; Erysipelas phlyctænodes; (*Of H. zoster*) the Shingles; la Zone; la Sangle; le Ceinturon; der Gürter; Zostera; Zona repens; Circinus Cingulum; Zona serpiginosa; Zona volatica.

Definition.—Herpes (ἕρπω, to creep or spread), is an acute eruption of comparatively large vesicles which occur in clusters, each cluster being situated on a circumscribed patch of erythematous skin, and separated from neighbouring clusters by intervals of sound skin.

Causes.—Herpes *zoster* occurs at all ages. It is commoner in the summer than in the winter. It follows sometimes exposure to cold, sometimes violent fits of passion. At certain irregular periods, cases of H. zoster get suddenly to be very much commoner than they usually are, and an epidemic of the disease, if it may be called so, prevails.

Herpes *labialis* sometimes occurs as an independent complaint, affecting the lining membrane of the mouth and fauces, as well as the exterior of the lips, and attended with slight febrile disturbance; but ordinarily it is an accessory to, and appears in the course of other diseases, such as catarrh, ague, continued fever. Herpes *præputialis* is almost peculiar to adults; it is often associated with stricture of the urethra; by some authors of weight, however, this is regarded as a mere coincidence.

Description.—The varieties of Herpes are H. zoster, H. phlyctænodes, H. labialis, H. præputialis, H. circinatus, and H. iris.

Of these the two last are parasitic diseases, and will be treated of accordingly with other eruptions of that group. *Herpes zoster* or Zona is an acute affection which begins by slight febrile disturbance, a sense of weariness, pain in the loins, and loss of appetite. Then, over a limited region, sensations of heat, or of tingling, or even darting pains may be felt. Shortly, on the region so affected, appear several circumscribed erythematous patches of irregular shape. These are quite distinct from one another, and vary in size from half an inch to three or four inches across. Their general arrangement is such that they form a wavy interrupted band, which is situated generally on the trunk, where it takes a transverse direction, reaching half round the body on one side (usually the right), commencing at the middle line behind, and extending obliquely forwards and a little downwards as far as the middle line in front. Shortly after the patches have appeared they become studded with minute transparent vesicles of the size of millet-seeds, and averaging in number from half a dozen to a score on each patch. Most of these continue distinct from one another; but some of them, as they increase in size, become joined with others of the same cluster, so as to form large irregular bullæ. Those however that remain separate rarely exceed the size of a pea.

In either case they soon cease to be transparent, but, retaining their brilliancy, assume a lustrous opaline appearance. This lasts for a day or two, when, about the fifth day of the eruption, the contained fluid gets turbid and inspissated, so that the vesicles become dull, flaccid, and opaque; and, at the same time, the erythematous patch on which they are seated begins to fade. The shrivelled blebs speedily dry up into small brown crusts, which conceal superficial excoriations; the latter soon heal, and the crusts fall off, leaving red stains, which gradually fade away. The number of the groups of Herpes is very variable; the half-belt may either consist of a good many, or be made up of only two or three. The development of the different clusters is not simultaneous; they appear in succession, so that at any given time, during the progress of the eruption, different patches will exhibit different stages of development or decline; vesicles of the same cluster, however, always progress *pari passu*.

The course of the disease is usually completed in from ten days to a fortnight; sometimes, however, it is more protracted: either the vesicles may get accidentally ruptured, and so leave behind them superficial ulcers, which necessarily prolong the disease, or in the aged or cachectic a grey slough (H. gangrenosus) may form beneath and around the vesicle, and leave after its separation a deep painful ulcer, which may take even several months to heal. This condition of the skin is attended generally with hectic fever and great prostration, and the sores leave behind them permanent scars; or, towards the termination of the eruption, more or less severe lancinating neuralgic pains may attack the region occupied by it. These pains are more intense, and are prolonged over a greater length of time in the aged and debilitated than in the young and vigorous. Their duration is usually limited to a few days, but they may continue for several weeks, months, or even years, after the disappearance of the eruption. Zona is almost invariably confined to one side of the body, stopping short both behind and before at the median line, which it rarely transgresses. It has in a few instances been observed on both sides of the trunk, but on these occasions the two halves of the belt did not correspond, the one being situated considerably above the level of the other. (There is a popular notion, which however is without the least foundation, that such cases necessarily prove fatal.) It appears usually on the trunk, the lower part of the thorax being its most favourite locality; but it occurs also on the neck, the face, the head, and the upper and lower limbs. On the limbs its direction is vertical; on the trunk, horizontal; and on the neck, face, and head, partly vertical and partly horizontal. On the trunk the vesicles of Herpes are larger than in the head, neck, or limbs. *H. phlyctænodes* is a division of Herpes that was established on the assumption that the variety just described varied in essential particulars accordingly as it occupied the trunk or other parts, and comprehended all cases of H. zoster occurring elsewhere than on the trunk. *H. labialis* occurs, as its name implies, about the lips. In the place where it is about to appear a sensation of burning and smarting is felt; this is followed slowly by the appearance of a circumscribed red patch, which soon becomes swollen and shining. On this a cluster of small vesicles is speedily developed; some of these may unite to form a small irregular bleb of about the size of a split-pea. The colourless transparent vesicles soon become yellow and opaque, and the burning and smarting sensations subside; the vesicles dry up into small brown crusts, which fall about the seventh or eighth day, leaving red stains, which gradually disappear. H. labialis appears generally at the junction of the mucous membrane with the skin, but often affects the skin at some little distance from the red margin of the lips. It is common at the angles of the mouth, but it may be situated over any part, either of the upper or lower lip; sometimes it forms a complete circle round the mouth. It occasionally appears on the mucous membrane lining, the buccal cavity; in this situation the vesicles soon rupture, and are replaced by little white patches of macerated epithelium. *H. præputialis* may affect either the outer or inner surface of the prepuce, appearing in the form of small, red patches. Each of these is about the size of a fourpenny-piece, is perfectly distinct from the others, and soon becomes covered with a crop of transparent globular vesicles. The eruption is preceded and accompanied by itching, heat, and sometimes smarting of the prepuce. When it affects the outer surface of the foreskin, the vesicles soon become opaque, shrivel, and are replaced by small crusts, which fall about the seventh or eighth day. On the inner surface of the prepuce, the affection is attended with more irritation, and the patches are redder; the vesicles, which soon burst, are replaced by little superficial ulcerations that speedily heal. Sometimes, however, in this situation, the disease is kept up by a succession of eruptions, and assumes a chronic character. The end of the prepuce becomes thickened, wrinkled, and even fissured, and at the same time the margin of its orifice gets gradually contracted and converted into a ring of almost cartilaginous hardness.

Diagnosis.—H. zoster may be mistaken for Erysipelas, Eczema, or Pemphigus; H. labialis for Eczema; and H. præputialis for Chancroid. However, in *bullous Erysipelas*, the inflamed areola is generally much more extensive than in Zona. On the other hand, the blebs are much less numerous and a good deal larger, besides being irregular in shape; the margin of the erysipelatous surface, too, is distinctly raised. In *Eczema*, although that disease may occur in patches, yet the patches have neither the well-defined margins nor the systematic arrangement of Zona. Again, the vesicles of Eczema are smaller and much more crowded together than those of Herpes. The isolation and the volume of the vesicles of Herpes may cause it to be mistaken for *Pemphigus;* but in the latter, although the bullæ may be small and near to one another, they are never arranged in systematic groups. Zona is an acute, Pemphigus almost always a chronic disease.

From H. labialis *Eczema* may be readily distinguished by the absence of a definite margin, and the minuteness and agglomeration of the vesicles.

H. præputialis is very liable to be mistaken for Chancroid; but the former disease begins as a cluster of vesicles, the latter is almost from the commencement an ulcer. Later, the herpetic scales on the outside of the prepuce can scarcely be confounded with the black, thick crusts of syphilis; nor the herpetic excoriations of the inner prepuce for the chancroid ulcer, which has a yellowish floor, and however shallow it may be, has always abrupt edges.

Prognosis.—In H. zoster, H. labialis, and H. præputialis, prompt recovery may usually be anticipated H. zoster, however, in old and debilitated persons, is apt to assume a gangrenous form, and to be succeeded by persistent neuralgia; and H. præputialis, when it affects the inner surface of the prepuce, will often assume a chronic character, and become an obstinate and troublesome affection.

Treatment.—Herpes zoster, when it runs its usual favourable course, requires but little treatment. If there be much febrile disturbance at the commencement, diluent drinks should be given, and a light diet enjoined. The bowels should be regulated, if necessary, with mild aperients, and (if the eruption be considerable), the patient be directed to remain quiet. In the employment of local remedies some caution should be exercised, for applications that would seem to be indicated by the acute inflammation of the skin, such as poultices, fomentations, and emollient lotions, macerate the walls of the vesicles, facilitate their rupture, and so, as has been seen, not only prolong the course of the disease, but add considerably to the discomfort attending it. The best lotion, if a lotion be used, is a solution of the acetate of lead; but it will generally be better to oil the surface with almond- or olive-oil, and then to dredge it over lightly with starch-powder. This plan is very efficacious in protecting the vesicles from rupture, and, in an uncomplicated case, to preserve them unruptured is the main object in view. When the vesicles have dried up, a few warm baths may be taken to facilitate the separation of the crusts. Should the vesicles become accidentally ruptured, the ulcers should be dressed with ung. plumbi subacet.; or, if painful, with an ointment made with moist extract of opium (a drachm to the ounce and a half of simple ointment). When the disease occurs in old or cachectic persons, their general health should be attended to. In the gangrenous form, such tonics as bark or quinine, stimulants, and a nutritious diet, should be prescribed; locally, stimulating lotions, or dusting the surface over with sulphate of quinine, are the best remedies.

When the eruption is complicated with neuralgic pains, an ointment of belladonna or aconite-liniment, containing to the ounce half a drachm of the former, or two drachms of the latter, may be employed locally, while the tincture of either of these plants is given internally. If there be much anæmia, the milder preparations of iron, in small doses, will be of service in relieving the pain. When the eruption has altogether disappeared, the application of blisters, and dusting the blistered surfaces over with small quantities of morphia, will often succeed in arresting the neuralgia. The hypodermic injection of a solution of a salt of morphia, gives speedy and often complete relief. The thermic hammer, the linimentum chloroformi et belladonnæ, moxas, and, in desperate cases, section of the affected nerve, are also valuable resources.

H. labialis is always a trivial affair, and requires scarcely any treatment. The application of such liquids as lead-lotion containing a little proof-spirit, glycerine, a mixture of a drachm of the liquor plumbi subacetatis with an ounce of fresh cream, &c., is all that is necessary.

H. præputialis, if acute, needs only the simplest treatment. When situated on the outside of the prepuce, it requires chiefly protection from the friction of the dress. When the inner surface of the fore-skin is affected, the urine should be rendered bland and unirritating by the administration of diluents and demulcents, and a piece of dry lint be introduced between the glans and prepuce to prevent contact of their surfaces, and to absorb irritating secretions. If there be much irritation, emollient lotions should be injected carefully beneath the prepuce, and the penis bathed frequently in warm water. In the chronic affection, citrine ointment, or carbonate of zinc ointment, is requisite; and Plummer's pills, or the alkaline sulphurets, should be administered internally. When the præputial orifice has got much contracted, operative interference becomes necessary.

SECTION V.—PUSTULÆ.

The pustular eruptions are Ecthyma and Impetigo.

ECTHYMA.

Synonyms.—Phlyzacia; Boutons; Epinyctis; Ecchymata; Echrasmata; Terminthus; Thyma.

Definition.—Ecthyma (*ἔκθῡμα*, a pustule) is characterised by an eruption of large, rounded, discrete, flattened pustules, resting on an inflamed base, and giving rise to the formation of a dark-coloured adherent crust, which, on separating, discloses a brownish- or purplish-red stain, which is slow to disappear.

Causes.—The predisposing are dirt, poverty, hunger, exposure to cold and moisture, prolonged mental excitement, excessive bodily fatigue, drunkenness, debauchery, watchfulness. The disease is consequently commoner with the poor, the aged, and the dissipated. It sometimes follows an attack of specific fever, *e.g.*, scarlet fever, measles, typhoid fever.

But, independently of these causes, it may be excited by the contact of local irritants, *e.g.*, tartar-emetic, sugar, lime, iron-filings; thus it is often seen on the hands and forearms of grocers, bricklayers, and workers in metal. There is another local irritant to which a large proportion of the cases of Ecthyma are due, viz., the acarus scabiei.

People in whom Ecthyma may thus be excited by mere local irritation are as a rule of the lymphatic temperament. The disease is not contagious, but it is inoculable.

Description.—Ecthyma may follow either an acute or a chronic course.

Acute Ecthyma is preceded by more or less feverishness, and (usually) by tingling, pricking, or smarting of the surface about to be affected. The eruption, which is confined to a limited area, appears at first in the form of elevated, rounded, red patches of the average size of a pea, and distinct from one another. These patches become speedily changed into lenticular pustules of corresponding size with red areolæ. After a few days the pustules dry up, and are replaced by hard, dark, thick crusts, which, on separating, disclose dusky-coloured red stains, which may last for a considerable time. Generally several crops of pustules appear during the course of the eruption, which extends usually over about ten days or a fortnight. This variety is seen more commonly on the limbs and neck than on the trunk or head. It is attended sometimes with considerable inflammation of the surrounding skin and of the neighbouring lymphatic glands, and not unfrequently is complicated with furunculi. This is the commonest variety of Ecthyma, and hence was called by Willan *E. vulgare.*

Chronic Ecthyma differs from the acute variety not only in duration, but also in extent. The later crops of pustules, instead of occupying the same region as their predecessors, invade successively fresh portions of skin. Again, the febrile disturbance which is associated with the eruption, instead of commencing a few hours before, and subsiding a few days after the appearance of the latter, assumes a hectic type, and becomes gradually more marked as the eruption progresses. This variety sometimes affects infants at the breast (*E. infantile*), who are reared under unfavourable hygienic circumstances. In infants it attacks chiefly the regions which are most exempt from the acute variety, viz., the face and chest. It is a serious affection, since it is apt to become complicated with diarrhœa and copious sweating, and in a large proportion of cases terminates fatally. Chronic Ecthyma occurs also in adults whose vitality has been lowered by dissipation, grief, privation, or old age (*E. cachecticum*). In this variety the pustules are larger and flatter, as well as more flaccid, than in either of the preceding; they are surrounded by a livid-red areola, and contain a dark-coloured sanious pus; they are replaced by hard, flat, black crusts from beneath which a dirty, ill-smelling discharge exudes; on detaching one of these crusts, a pale, flabby ulcer is disclosed. E. cachecticum affects especially the lower limbs; it may last indefinitely.

Diagnosis.—The diseases with which Ecthyma is most likely to be confounded are Rupia, Impetigo, Acne, Furunculi, and pustular Syphilides.

Between *Rupia* and Ecthyma there is certainly a great analogy, and some authors of repute have classed them under one name. But in Rupia the elevation of the epidermis is broader, and the contents of the bleb, when recent, are serous. Again, the crusts of Rupia are stratified and are always decidedly thicker at the centre than at the circumference.

In *Impetigo* the pustules are much smaller, and much more numerous; they are clustered together and are often confluent; the crusts are more uneven, are lighter-coloured, softer, and moister, as well as more extensive than those of Ecthyma.

In *Acne* the pustules, which are small and acuminated, rest generally on a non-suppurating, elevated, indurated base; they are confined to the face, shoulders, or chest.

From the *Pustular Syphilide,* which is an occasional variety of infantile syphilis, infantile Ecthyma may be distinguished by the absence of mucous tubercles.

In *Syphilitic Rupia* the coppery areola around the bleb, the greenish tint of the crusts, the deep ulcer it covers, the long course run by each bleb, and the general history of the case, will serve for differential diagnosis.

Prognosis.—This will vary according to the cause of the disease: Thus, when the Ecthyma has been produced by local irritation, a speedy and complete recovery may be anticipated. So also with acute Ecthyma, at least in the majority of cases. In the cachectic variety the prognosis should be guarded. And in infantile Ecthyma a fatal result is to be apprehended.

Therapeutics.—Acute Ecthyma requires only very simple treatment. Refrigerant drinks, slight laxatives, and local emollient-baths, comprise all that is necessary. In chronic Ecthyma it is of the first importance to remove the patient from amongst the causes which have depraved his health.

In infantile Ecthyma a good wet-nurse is often essential to recovery. Scrupulous cleanliness, pure air, and regular repose, are of scarcely less importance. Cod-liver oil, and bark, and ammonia should be given internally, and the condition of the bowels should be carefully regulated. The sound as well as the affected skin should be powdered with a mixture of sulphate of quinine and lycopodium.

In *cachectic Ecthyma* change of air, especially removal to some suitable part of the sea-coast, light animal food, fresh (cooked) vegetables, and a moderate allowance of alcholic stimulant, should be enjoined. At first small doses of opium (gr. ½ ter quotidie) will be found of service. Afterwards ammonia, valerian, bark, quinine, and the preparations of iron, should be employed. Externally alkaline-, sulphurous-, or sea-water-baths should be used. The local applications should be stimulating, and the dressings should be as light and simple as possible. All relaxing applications, such as water-dressing covered with oil-silk, poultices, and, above all, plasters should be avoided.

IMPETIGO.

Synonyms.—Crusted tetter; Cowrap; Dartre crustacèe flavescente; Lepre humide; Nässende grind; Melitagra; Psydracia; Lichen vitiligo; Ecpyesis impetigo; Porrigo favosa; Crusta lactea.

Definition.—Impetigo (Impĕto, to attack) is an eruption of minute pustules set closely together and producing thick, moist, yellowish scales.

Causes.—The lymphatic temperament, hereditary transmission, dentition, impaired health, the seasons of spring and autumn, exposure of the skin to the contact of certain irritating substances, viz., sugar, lime, metal-filings, or of irritating secretions. The irritation produced by linseed poultices, or by certain parasites of the skin, viz., the acarus scabiei, the pediculus capitis, the achorion schönleinei.

Description.—Impetigo begins as an eruption of numerous minute yellowish pustules of the size of a pin's-head or that of a millet-seed, closely clustered together on a more or less reddened portion of the skin. In a day or two the pustules burst, and their opaque viscous contents exude and dry up into a yellowish crust, from beneath the edges of which a purulent discharge exudes. Since it is in this latter condition that the disease generally comes under observation, it is of more importance for the purposes of diagnosis to be acquainted with the characters of the scab than of the pustules which produce it.

The scabs of Impetigo form moist, yellowish, thick patches. Their surface is uneven and often nodulated. Their colour is a dull yellow, tinged often with green or brown. When a crust has lasted for some time, its surface becomes slightly lamellated, so as to give it a somewhat flaky appearance. When the disease is spreading, fresh pustules may be seen around the edge of the crust. On removing a portion of a crust, a raw, moist, suppurating surface of a pale pink colour is disclosed.

Varieties.—The principal are *I. figurata* and *I. sparsa*. In I. figurata a large number of pustules crowded together on a limited surface give rise to a circumscribed patch of the disease. In I. sparsa the pustules are scattered over a large extent of surface in small groups. When Impetigo figurata occurs on the whiskers, moustache, or beard, it is termed *I. sycosiformis*, from its resemblance to Sycosis. When it affects other portions of the face it is called *I. larvalis* (larva, a mask).

Impetigo sparsa of the hairy scalp, when it produces small, thick, dry, brownish scabs, is termed I. granulata. When the disease partakes of the character of I. figurata as regards the crowding of the pustules, but resembles I. sparsa in the extent of surface covered, so that, *e.g.*, the whole of the forearm or the whole of the leg becomes encased in a scab, it is called I. scabida. Impetigo is rarely preceded or attended by any febrile disturbance, or with any kind of local sensation. It occurs most commonly in children, affecting especially children of lymphatic temperament. When it occurs in adults it happens most frequently to the obese and flabby, or to those of broken-down constitution. It is in a large proportion of cases a constitutional disease, but it is not uncommonly a mere phase of Scabies. Very many of the cases of Impetigo of the scalp occurring in children are, according to the author's researches, entirely dependent on the irritation set up by the pediculus capitis; in such cases the occipital part of the scalp is almost invariably the part chiefly affected, the sincipital part of the scalp being almost, if not altogether, free from eruption; he has found the converse to be the rule in cases of constitutional Impetigo affecting the scalp. Again, the former appears most commonly as I. granulata, whereas the latter assumes usually the characters of I. figurata.

Diagnosis.—The diseases for which Impetigo is liable to be mistaken are Eczema, Ecthyma, Sycosis, Tinea favosa, pustular Lupus, and syphilitic Rupia. But in *Eczema* the discharge is transparent, the scabs are thin and lamellar, and in the early stage vesicles take the place of pustules. Notwithstanding these differences, however, there is between Eczema and Impetigo a very close analogy. In *Ecthyma* the pustules are large, few in number, and discrete, and the crusts small and dark-coloured. From *Sycosis*, I. sycosiformis may be distinguished by the lack of subcutaneous inflammation and induration, and by its not (until after very long continuance) producing local alopecia. From *Tinea favosa*, Impetigo of the scalp may be distinguished by the fact that, although the latter often produces more or less thinning of the hair, it never leads to extensive alopecia; again, its dull brownish-yellow crusts have but little resemblance to the bright sulphur-coloured cup-shaped incrustations of favus. The absence of deep ulceration and subsequent cicatrices, will distinguish Impetigo from *pustular Lupus* and from *Syphilitic Rupia.*

Prognosis.—This, as Impetigo never threatens life, is confined to the probable duration of the disease. An acute attack lasts usually two or three weeks. In the chronic state the disease may last several months. After recovery the disease is apt to recur. It is in scrofulous persons that the disease assumes its most chronic and obstinate phase, and is most apt to reappear.

Treatment.—In acute Impetigo the diet should be restricted; laxative and refrigerant medicines should be administered; and the local applications should be emollients, such as warm decoctions of marshmallow or of poppies, infusion of linseed or almond-mixture, or poultices of bread or of ground rice, followed by light dressings of oxide of zinc or acetate of lead ointment.

In chronic Impetigo the most efficient internal remedies are cod-liver oil, the various preparations of iron, bitter tonics, the sulphurous mineral-waters, and occasional laxatives. Externally, ointments of precipitated sulphur, of the nitrate, the oxide, or the bichloride of mercury, or the huile de Cade, or lotions of alum, tannin, chloride of zinc, or hepar sulphuris, are the best applications. When the purulent discharge is thin and profuse, a mixture of tannin and starch in suitable proportions should be dusted over the part.

In all cases of Impetigo of the scalp the head should be shaved, or at least the hair should be cut close. When the occipital region of the scalp is the part chiefly affected, the application of ammonio-chloride of mercury ointment should form a part of the treatment.

SECTION VI.—BULLÆ.

The bullous eruptions are Pemphigus and Rupia.

PEMPHIGUS.

Synonyms.—Water-blebs; Blister tetter; Dartre bulleuse; Angine pemphigöide; Blasenausschlag; Pompholix; Morbus vesicularis; Phlycteme; Hydroexanthema bullosum; Morbus phlyctenöides.

Definition.—Pemphigus (πεμφιξ, a bleb) is a disease characterised by the development of a greater or less number of clear watery blebs on inflamed red surfaces; these little bladders, being readily ruptured, give rise at first to excoriations, and afterwards, by the concretion of their liquid contents, to thin crusts.

Causes.—Exposure to cold and moisture; bad or insufficient food; mental distress, (rarely) pregnancy. The debility that is associated with chronic Pemphigus is sometimes the cause and sometimes the effect of the disease.

Description.—Pemphigus may follow either an acute or a chronic course.

Acute—a much rarer affection than chronic—Pemphigus is of three kinds.

In the one (*P. solitarius*) an extremely rare variety, which is seen only in old persons, there appears, usually on the shank, a single bulla of about the size of a nut; this rapidly increases in size. When fully formed it produces a painful feeling of tension. After remaining stationary for a day or two, the bulla becomes flaccid, and at the expiration of a week disappears; a second and sometimes a third bulla may appear near the situation of the first. The appearance of each bleb is preceded by burning and itching, and sometimes by redness.

In another variety (*acute general Pemphigus*), less rare than the preceding, the eruption, preceded for a day or two by febrile disturbance, appears in the form of small circular rose-red patches. In the course of a few hours the centre of each patch becomes opalescent and somewhat raised, and in a short time a transparent bleb, surrounded by an erythematous ring, is developed; in a few days the bulla attains its full growth. Its production is accompanied by itching and tingling. Its manner of disappearing is not in all cases the same. In some instances the serum is gradually reabsorbed, so that the bulla shrivels, and is replaced by a dry foliaceous desquamation; but more commonly the bleb bursts and the serum escapes, disclosing a more or less extensive excoriation. This may either get covered by a thin dark-coloured crust, which after a time falls off, or the epidermis becomes reapplied to the excoriated surface, which heals under it. In either case a violet stain is left, which may last for some time. The duration of each bulla is about a week, but by a succession of several crops of bullæ, the disease is generally prolonged for about three weeks or a month.

The remaining variety of acute Pemphigus is peculiar to new-born infants, and affects only the palms of the hands and the soles of the feet. The blebs, which contain a transparent yellow serum, vary in size from that of a pea to that of a small bean; each of them is encircled with a dusky violet-coloured areola. They soon rupture and get replaced by superficial ulcers, which become covered by thin black crusts. The ulcers spread by the formation of fresh bullæ. This variety is supposed by some authors of weight to be a form of infantile syphilis. It is accompanied by sickness, diarrhœa, and marasmus, and is apt to terminate fatally.

Chronic Pemphigus, in its ordinary form, may either succeed to acute general Pemphigus, or may from the first maintain a chronic character. It differs from the latter disease in being preceded by only slight febrile disturbance, in the absence of a red areola around the bullæ, which moreover are less tense and transparent than in the acute variety, and in the more chronic character of the ulcers left after the blebs. It resembles the acute disease in producing a succession of crops of bullæ. These may follow one another at intervals of only a few days, or may be separated by such a pause as to induce the belief that permanent recovery has taken place. When so long a term intervenes, the succeeding crop assumes much of the character of an acute attack.

Sometimes the variety just described may lead to another more serious condition (*foliaceous Pemphigus*). The bullæ increase in number, become confluent, and appear in quicker succession, till at last they become so crowded and succeed one another so rapidly that they are unable to go through the usual phases. They have scarcely formed when they rupture, and leave the surface covered with little lamellar crusts, which resemble pretty closely in colour, consistence, and thinness, the layers of a piece of light pastry. These flakes, which adhere but loosely to the skin, being curled up much in the same way as the loose bark of a sycamore tree, are readily detached and as readily reproduced. The skin itself is moist, exuding a fœtid, nauseating, serous discharge. This foliaceous condition of the skin is apt to invade the whole surface.

Pemphigus, although a comparatively rare, is always a grave affection in the acute form, on account of its tendency to recur and finally pas into the chronic state; and in the chronic form on account of th debility and emaciation that are induced by it. Pemphigus may occu at any age, and may appear at any part of the surface. In the chroni condition it is apt to be complicated with obstinate diarrhœa. It i sometimes attended with severe itching (pruriginous Pemphigus).

Diagnosis.—Pemphigus may be mistaken for Herpes Zoster, Rupi Ecthyma, Impetigo, or Erysipelas.

Herpes zoster may be distinguished by its tendency to form a hal zone round the trunk by the smaller size of its blebs, which rarel exceeds that of a pea, and by the severe neuralgic pain that ofte accompanies it.

In *Rupia* the bullæ are fewer in number, are flatter, and are les commonly arranged in groups than those of Pemphigus; the crusts too, are thicker and are conical, and rest upon actual ulcers.

In *Ecthyma* the liquid that raises the epidermis is always purulent and the crusts are black and thick.

The crusts of *Impetigo* may always be distinguished from those o Pemphigus by their greater thickness, their yellow colour, and thei brittleness.

In *Erysipelas* the blebs are irregular in outline and are flatter, and the intervening skin is shining, swollen, and inflamed.

Prognosis.—In acute Pemphigus (excepting P. neonatorum) th probability as regards the individual attack is always in favour o recovery, but a relapse is to apprehended.

When the disease is chronic, the prognosis should be guarded o account of the intractable nature of the disease, and its tendency when it has become extensive and has assumed the foliaceous condition, to terminate fatally.

In P. neonatorum the prognosis is grave.

Treatment.—Acute general Pemphigus should be treated by diluen drinks, by laxatives, and by emollient applications. Care should b taken to avoid rupturing the bullæ, or, should they have burst, t prevent the epidermis getting rubbed off from the raw surface protected by it; with this view rest should be enjoined, and the affecte skin may be dusted over with "violet-powder." Any raw surfac should be dressed with some soothing and gently-astringent ointment *e.g.*, ung. zinci. oxid. P. neonatorum, P. solitarius, and chronic Pemphigus, all require the same general treatment. A generous diet, moderate allowance of wine, tonics—quinine, iron, arsenic, and small opiates. As regards the local treatment, the blebs, unless of considerabl size, should not be punctured. The raw surface left after the ruptur of a bleb should be dressed with ointment or lotion of the acetate o lead; or, if the ulcer exhibit a tendency to take on a chronic condition, with a lotion of the nitrate of silver. When there is much tenderness and pain, the ung. gallæ c̄ opio. is an excellent application.

RUPIA.

Synonym.—Ulcus atonicum.

Definition.—Rupia (ῥυπέω, to be foul and filthy) is an eruption of large, flat, discrete bullæ, which contain at first a serous, and afterwards a purulent fluid, and which produce dark crusts, thicker at the centre than at the circumference, covering shallow ulcers.

Causes.—Insufficient or unwholesome food, poverty, dirt, privation, damp habitation, an unhealthy atmosphere, drunkenness, old age, a recent attack of scarlatina or of smallpox.

Description.—In its mildest form (*R. simplex*) the disease appears in the form of small, rounded, but flat blebs, the diameters of which vary from that of a threepenny-piece to that of a shilling. These are moderately distended by a thin, opalescent, serous liquid, which soon gets purulent, and at the same time inspissated, so that the blebs become flaccid, and finally dry up into uneven brown crusts which are thicker at the centre than at the circumference. These crusts are readily detached, and leave superficial indolent ulcers, which finally cicatrise, leaving behind them livid red stains, which may persist for some time. While these changes are taking place in the first crop, fresh bullæ continue to be formed, which follow the same course, the disease lasting altogether about a fortnight or three weeks. This variety approaches very closely in its characters to Ecthyma, and is considered by several authors of repute to be identical with it.

In a severer form (*R. prominens*) the disease commences as small rounded, inflamed spots, on which bullæ become speedily developed. The serum contained in these is often dark-coloured and sanious, and is usually of thicker consistence than in the preceding variety. The blebs are replaced by thick dark-coloured crusts, which are surrounded by a dusky erythematous areola. This areola soon becomes converted into an annular purulent bleb, which in its turn becomes converted into a scab. In this manner the crust, by successive additions at its circumference and under-surface, acquires a conical shape. In some cases the growth at the under-surface makes greater progress than that at the circumference, and the crust acquires the appearance of a limpet-shell; in others the growth at the circumference is more rapid, and then the crust is flatter, and has much of the form as well as the laminated appearance of an oyster-shell. In either case the crusts are generally dry, hard, dark-coloured, and pretty adherent, so as to acquire often a considerable thickness; when detached, a pale, spongy, ragged ulcer is disclosed, which bleeds on the slightest touch, and is deep in proportion to the duration of the crust; this slowly cicatrises and leaves a purplish stain, which remains for some time.

R. escharotica is a variety still more severe; it attacks sickly infants, commencing by livid-red patches, on which bullæ, containing a dark sanious fluid, become developed, the bullæ spread, and instead of forming crusts, leave open foul, ragged sores, with livid, raised edges.

These sores are very slow to heal, and are moreover extremely painful so as to prevent rest; they are generally attended with fever; severa of them appear in succession.

Rupia attacks preferably the loins, nates, and lower limbs, bu occurs also on other parts of the body. It may appear at any perio of life, but is commoner in childhood and old age.

Diagnosis.—Rupia is liable to be mistaken for Ecthyma, *Syphiliti* Rupia, and Pemphigus.

In *Ecthyma*, although it originates in similar causes and bears a considerable resemblance to Rupia, yet the crusts have not the conical formation, nor are they accompanied by the decided ulceration produce by the latter disease.

Syphilitic Rupia, again, closely resembles the simple disease, but it crusts are even blacker and harder, and have often a greenish tinge they cover much deeper ulcers, the floors of which are covered with grey pultaceous substance, and the edges of which are perpendicular the areolæ around them are coppery instead of livid, and there ar always other syphilitic symptoms to aid the diagnosis.

Prognosis.—With the exception of R. escharotica, Rupia is not i itself dangerous to life. When death occurs in the course of it, th result is due to the general state of health which caused the disease and not to the disease itself; recovery, however, always takes plac slowly.

Treatment.—It is of the greatest importance to improve the patient' general health, and to remove the causes which have depraved i Light, easily-assimilable animal food and fresh vegetables, an occasional laxative, small opiates, bitter tonics, chalybeates, good malt liquors, wine, cod-liver oil, and mineral acids, are the most importan internal remedies.

Externally, tepid, alkaline, or sea-water baths, and gently-stimulating local applications, aided by an occasional pencilling with nitrate o silver, are useful. When the legs are affected with Rupia, the recumben posture, with the legs somewhat raised, greatly facilitates recovery.

In obstinate cases a sea-voyage is much to be recommended. I R. escharotica, to support the strength and allay pain, and locally t employ anodynes, antiseptics, and emollients, are the chief maxims.

SECTION VII.—TUBERCULA.

The tuberculous eruptions are Lupus, Cheloid, Verruca, Acne, an Molluscum.

LUPUS.

Synonyms.—The Wolf; Corroding tetter; Esthiomène; Dartre rongeante; Dartre vive; Dartre ulcérée; Dartre phagédénique; Fressend Flechte; Herpes exedens; Herpes ferox; Herpes serpiginosus Darta maligna; Lupus vorax.

Definition.—Lupus may be defined as an extremely chronic disease of the skin, affecting young persons, associated with the lymphatic temperament, and often with scrofula, characterised at first by purplish redness and more or less swelling of the affected skin, and terminating in the formation of a permanent cicatrix with or without previous ulceration.

Causes (predisposing).—The lymphatic temperament; the scrofulous diathesis; childhood; the female sex; hereditary transmission; unhealthy habitation; unwholesome or insufficient food; a moist atmosphere; confinement.

(Exciting).—Sudden alternations of heat and cold; local irritation of various kinds.

Of these causes the most active are temperament, diathesis, and age. Lupus may be said to have invariably associated with it a lymphatic temperament, and it is evidently closely allied to, if not identical with, scrofula. Although owing to the great length of time over which its course extends it happens to be met with in middle-aged and even in elderly persons, yet its *first appearance* dates in most cases from childhood, and it is extremely rare for it to appear after adult age has been attained. Sex has not much influence in its development, still it is somewhat commoner in females than in males. Hereditary transmission has but little influence in its production, except in so far as it affects temperament and diathesis. The remaining causes that have been enumerated as predisposing to lupus are only accessory to the preceding.

Description.—Lupus is met with most frequently on the face, affecting usually the nose or the cheek, but it occurs also on other parts of the body.

There are three varieties of this disease, viz., the erythematous, the pustular, and the tubercular.

Erythematous Lupus commences as a rounded, slightly-elevated, shining, lakish-red patch, not larger than a fourpenny-piece; on this after a while desquamation takes place. The scales that appear are white and opaque, and are arranged often in thin, curved, wavy, bands, which adhere firmly to the skin beneath. The patch spreads very slowly at the circumference, while the centre heals and becomes a thin, polished, slightly-depressed, white, indelible scar. This cicatrization takes place without any preceding ulceration, and is the effect of interstitial absorption.

Pustular Lupus begins, like the preceding variety, as a somewhat raised lakish or livid patch. On this shortly appear a few minute pustules of the size of a pin's-head, or in some cases one or two rupial blebs of the size of a split-pea. After about a week's duration they rupture and exude a plastic liquid, which concretes into a white, a yellowish, or a drab-brown crust. This crust, like the scales of the erythematous variety, adheres pretty firmly to the skin beneath it. On detaching it with the finger-nail, it is seen to cover a ragged shallow ulcer, the floor of which is formed of pale-pink flabby granulations.

Around the crust the disease slowly spreads by the extension of t livid elevation and the formation of fresh pustules or mattery bleb which in their turn give place to crusts, while after a certain time t crusts at the centre fall off and disclose a white cicatrix which is mo depressed, more opaque, thicker, and more uneven than the scar the preceding variety.

Tubercular Lupus at its commencement occurs as a cluster of abo half-a-dozen small, rounded, dusky-red, soft, elastic elevations, of size varying from that of a hemp-seed to that of a pea; they ma be either distinct from one another, or agglomerated. They ar generally accompanied by more or less infiltration of the cellular tissu beneath and around them.

Lupus hypertrophicus.—This is sometimes so considerable as t cause the disease to resemble Erysipelas.

The patch slowly extends by the formation of fresh tubercle around the original cluster.

Tubercular Lupus may follow either of the following courses :—

It may terminate in cicatrization without previous ulceration.

Or superficial ulceration may take place, as in the case of pustula Lupus.

Or deep ulcers may be formed (*Lupus exedens*). The cicatrix left b these, when they have healed, is thicker, more opaque, and more uneve than that of any of the preceding varieties. It resembles very close the puckered scar of a severe burn, and has the same property gradually but rigidly contracting, so as to occasion the most seriou deformities.

Or (lastly) the granulations that form the floor of the ulcers, pro duced either by pustular or tubercular Lupus, may become hard an dry, and acquire a thick tough layer of epithelium (*warty Lupus* This (warty) condition is succeeded, like all the varieties of Lupus, b a permanent scar, with or without previous ulceration.

Lupus in any of its forms is a comparatively rare disease. It is, a rule, unattended either with itching or "burning."

In some cases, although they are comparatively rare, a long slantin patch of Lupus will eventually become the seat of a well-marke epithelial cancer.

Prognosis.—Although Lupus in no way endangers the life, or eve the health of the subject of it, and may even, in cases of long stand ing, coincide with excellent general health, yet its proneness to attac the face, the ravages that, if unchecked, it commits there, it tenacity when it has once gained a footing, and its obstinacy unde treatment, render it a very serious affection. When it attacks the ey lids it is apt to affect both the outer and the inner (conjunctival surface of the lid, and in such case, if the disease happen to be othe than of the most superficial kind, it meets from opposite surface in the cartilage of the lid, and the result is the total destructio of the lid, a constant flow of tears over the cheek, and the establishmen of obstinate granular ophthalmia, followed sometimes by opacit and ulceration of the cornea, staphyloma, and permanent loss of vision.

When the nose is affected the same kind of result is apt to ensue; the disease creeps equally up the mucous and the cutaneous surface of the nostrils, and the result is often the total destruction of the cartilaginous portion of the nose.

So again, when the disease attacks the mouth, the face is apt to become much deformed by the disappearance of a greater or less portion of one or other of the lips. The external ear is apt to suffer in the same way.

Nor is it necessary that the disease should attack some one of the above-mentioned parts in order to produce considerable deformity. When the cheek alone is affected, if the ulceration have extended at all deeply, the gradual but irresistible contraction of the scar that is produced draws down and everts the lower eyelid of that side, so that the eye can no longer be completely closed; but besides this, the scar occasions considerable distortion of the features by drawing up the skin from other directions as well.

When the ulceration extends deeply it spares nothing: bone is as readily destroyed as muscle.

The prognosis, both as regards the extent of deformity likely to be produced, and the prospect of speedy recovery, will be far more favourable in the erythematous variety and in cases of tubercular Lupus, which exhibit a disposition to cicatrize without ulceration taking place, than in cases either of tubercular or pustular Lupus in which ulceration has taken place. The existence of much puffy infiltration of the subjacent cellular tissue is to be regarded as an unfavourable prognostic symptom.

The longer the disease has already lasted, and the more pronounced the scrofulous appearance of the patient, the more unfavourable will be the prognosis.

The most prolonged case of Lupus I have met with was in a lady a little over fifty years of age, who had been affected with the disease ever since she was three months old. It is common for Lupus to endure for ten or a dozen years.

Diagnosis.—Erythematous Lupus may be mistaken for Erythema papulatum, for Pityriasis, for Psoriasis, for Herpes circinatus, or for a Syphilide; but in Erythema papulatum the eruption has a much shorter duration; the patches are numerous, the limbs are affected.

In Pityriasis the skin is rough, the scurf is bran-like and not firmly adherent to the reddened surface, and there is usually itching.

In Psoriasis the scales are nacreous, are thicker, are stratified. The skin is harsh, or if smooth (Eczematous Psoriasis), is moist. The patches are usually numerous and widely spread, and if the eruption appears on the face, the eyebrows generally are chiefly affected. If the patches of Psoriasis be ringed there will be no cicatrix in the centre of the ring. Whether ringed or nummular, Psoriasis usually occasions itching.

In Herpes circinatus, the reddened skin is of a pink colour, is roug and harsh, and is far from being shining. The patches increase rapidl and there are generally several of them. The centre of each of th patches, although less raised and somewhat paler than the circu ference, offers no resemblance to a cicatrix.

From any similar syphilitic patch, erythematous Lupus may distinguished by its limited extent, the extreme slowness of its pr gress, and the absence of any other signs of constitutional Syphilis.

Pustular Lupus might be taken for Impetigo, but the latter erupti progresses far more rapidly, the duration of its pustules is mu briefer, the crust that succeeds them is of a lighter colour and soft consistence, and is less firmly adherent to the skin beneath it; th surface covered by the crust is not an ulcer. The eruption is usual more extensive, and less abruptly defined.

Tubercular Lupus, in its earlier stages, may be confounded wi tubercular Syphilide, especially with the clustered, non-ulceratin kind; but in the latter eruption, the tubercles are firmer an tawnier, and begin to ulcerate much sooner, there is no puffy infiltra tion of the cellular tissue beneath and around them, and there a usually with them other symptoms of constitutional Syphilis.

In the ulcerated condititition either pustular or tubercular Lupu may be confounded with an ulcerating tertiary Syphilide; bu whereas the syphilitic ulcer has even perpendicular walls, as if it ha been cut out with a punch, and a grey slough-like floor, and moreover surrounded by a coppery-red areola, the ulcer of Lupu has irregular scolloped walls, which are often undermined, so tha they overhang the circumference of the floor of the ulcer; the floo of the ulcer is composed of pale-pink flabby granulations, and th margin of the ulcer is surrounded by a purple-red areola.

The ulcer of Lupus may be taken for an ulcerating epithelia cancer, but the latter disease commences commonly in old age. Th ulcer produced by it is elevated at its circumference very considerabl above the level of the surrounding skin, and this raised circumferentia portion of the ulcer is distinctly lobulated.

Treatment.—The treatment of Lupus should include hygieni measures, general treatment, and local applications.

Regarding the *hygiene* of Lupus, attention should be paid to die climate, and exercise.

The diet should consist chiefly of mutton, beef, or game, eithe roasted or broiled, well seasoned with condiments, and relieved with suitable proportion of fresh vegetables, especially of those whic belong to the order cruciferæ, *e.g.*, cress, water-cress, radishes, & Coffee is a more suitable beverage than tea. A glass or two of win should be taken every day.

The patient should reside at some part of the sea-coast where th climate is dry and bracing, should be as much as possible in the ope air, and should take regular and active exercise.

The *general treatment* embraces the use of both internal and extern remedies.

The internal remedies employed are cod-liver oil, the preparations of iron, more especially the iodide, iodide of potassium, the liquor arsenicalis, Donovan's solution, the iodide of mercury, quinine, the various bitter infusions, more especially those of gentian, hop, or of the leaves of the walnut-tree, sea-water, &c.

Externally: baths, containing sulphuret of potassium, iodine, carbonate of soda, sea-salt or sea-weed, are made use of.

The *local remedies* are poultices and emollient applications to remove the crusts, huile de Cade, a solution of tannin in glycerine, the tincture of iodine, either alone or mixed with an equal quantity of the linimentum iodine, cashew-nut oil, ointments of iodine, of iodide of potassium or of iodide of lead, creosote-ointment, ointment of iodide of arsenic or of either the green or the red iodide of mercury, chloride of zinc, Vienna paste (equal parts of potash and lime), terchloride of gold, arsenic, sulphate of copper, nitrate of silver in powder or solution, butter of antimony, the actual cautery, carbolic acid, sesquichloride of iron, acid nitrate of mercury, caustic potash, chromic acid, &c.

Within the last few years, however, the treatment of Lupus has been successfully achieved in a comparatively rapid manner, by the process (so-called) of "erasion," namely, by scraping away the friable diseased tissue by means of a small steel spoon, and allowing the raw surface thus produced to granulate healthily. This process, however, is of avail only in dealing with thoroughly diseased areas of skin. The portions of skin in which the Lupus deposit is only scantily disseminated are reserved (after the performance of erasion) for treatment by "multiple scarification," which may be either of the "punctiform" or the "linear" kind.

KELOID.

Synonyms.—Cheloïde; Tubercules durs; Dartre de graisse; Le Crabe; Cancroïde; Cancre blanc; Cancelli; Kelos; Cancroma.

Definition.—Keloid is a small indolent persistent tumour developed in the thickness of the skin. It may be either pale, shining, oval, and flattened, or red, wrinkled, elongated, and prominent.

Causes.—The causes of keloid are very obscure. It is often developed on a cicatrix, but it appears also on perfectly sound skin. It has been known to follow a bruise. It is not hereditary, nor contagious, nor is it connected with any disturbance of the general health, or any specified constitutional condition.

Description.—Keloid is of two kinds, the one spontaneous or oval or "true," and the other cicatricial or cylindrical or "false" Keloid. *Spontaneous Keloid* occurs generally as a single tumour, which is developed usually on the front of the chest about the middle line, crossing the sternum transversely. Its general outline is oval. Its surface flattened, sometimes even slightly depressed towards the centre. Its consistence hard and resisting. It is pretty firmly rooted and immovable. When of long standing it is often extremely tender to the touch. The skin covering it has a tense shining appearance. Its colour, generally white, is sometimes pink.

At its margin the tumour gives off a number of filamentous digitations; these subside into the surrounding skin, which becomes somewhat puckered by their gradual contraction. The dimensions of the swelling rarely exceed two or three inches in length by a quarter of an inch in height. Often varicose venous radicles may be seen marbling its surface, and occasionally ligamentous-looking bands may be seen (through the tense skin) crossing the tumour near the surface. Spontaneous Keloid commences as a minute, hard, shining tubercular elevation, which takes several years to attain the size mentioned above. Its development is attended usually with itching, pricking, or even lancinating pain. It has been noticed on various parts of the body, but it occupies generally the chest, arms, or neck. *Cicatricial Keloid* is generally multiple. It presents nothing of the oval outline or flattened shape of the spontaneous variety. It appears as elongated prominent elevations of a semi-cylindrical shape, which bifurcate as they extend. They have some sort of resemblance to the larger divisions of the root of a tree where these appear half above the ground, and this resemblance is increased by the appearanee of the skin covering them, which is dry and wrinkled transversely to their length. Their colour is a tolerably deep red. This variety is less persistent than the other, and will sometimes disappear spontaneously within a few months after its commencement.

Prognosis.—Spontaneous Keloid once developed is apt to continue; sometimes, however, although rarely, its colour may become altered and the swelling subside, but some traces of it always remain. The cicatricial variety generally disappears completely of itself. The disappearance or diminution of Keloid tumours is effected by interstitial absorption; they have no tendency to ulceration. Neither variety of Keloid exerts any perceptible influence on the general health.

Diagnosis.—The disease cannot well be mistaken for any other affection.

Treatment.—Chloride of ammonium, cod-liver oil, or the alkaline iodides internally; ointments of iodine or of various iodides locally. Sometimes stimulating mercurial applications are of service. Severer measures, such as cauterization or excision, are at best useless, for, as a rule, even complete removal or destruction of the Keloid produces only a wound which heals with the formation of a still larger Keloid than before.

VERRUCA.

Synonyms.—Warts; Verrues; Porreaux; Warze; Verruca vulgaris; Thymus; Myrmecion; Lophos; Ficos; Muron; Acrochordon; Acrothymium.

Definition.—Warts are little tumours of the skin varying from the sixteenth to the eighth of an inch in height, which consist of a greater or less number of the natural papillæ of the skin, greatly hypertrophied, embedded in a mass of thickened cuticle.

Causes.—Although sometimes congenital they may commence at any age. It is far more common however for them to appear in children than in adults, and in the former case they are more numerous, although usually of briefer duration than in the latter. Thus, in young children it is not uncommon for a widely-distributed eruption of warts to disappear in the course of a few weeks. It is said that warts are contagious, and that they are more common on the hands of agricultural labourers and other people whose occupation entails the frequent handling of rough substances; with these statements, however, the author's experience does not accord.

Description.—Warts appear commonly as small tubercular elevations of the colour of the surrounding skin, which are somewhat constricted at the base. Their upper surface, which is rather more prominent at the centre than at the sides, is rough, rigid, and nodulated, the nodules being separated by linear depressions, so that the growth has something of the appearance of the head of a cauliflower in miniature. On attempting to detach a portion of a wart by the finger-nail, it is found that its substance may be readily picked off to a certain depth, coming away in the shape of nodules of epidermis, without either pain or bleeding being caused by its removal. If the top of a wart be sliced off, the section exhibits a reticulated marking, which divides the area into a number of polygonal spaces corresponding in size to the nodules seen on the free surface; in the centre of each of these spaces, if the slice removed be a thick one, is a small bleeding point. The wart so mutilated, if left to itself, soon regains its original size and appearance. The shallow linear depressions which have been referred to as mapping out the surface of the wart into nodules are sometimes extended as fissures deeply into its substance, so as to split it into segments, which may be made to separate temporarily from one another by pinching up between the finger and thumb the portion of skin on which the wart is situated. Sometimes, however, the segments, instead of being parallel and close-set, divaricate, so as to give the wart more of the arrangement of a bunch of carrots (*Verruca digitata*) than of cauliflower-head.

Warts, although their height is pretty constant within the measurements before stated, may vary considerably in area; this may be so limited that the wart has a pedunculated rather than a sessile appearance, its constricted base giving it the shape of a club; or the area may be so considerable as to give it the appearance of an elevated patch more than of a tubercle; sometimes the patch assumes the shape of a wavy band. Warts may appear on any part of the surface, but they are especially frequent on the face, neck, and hands. They are so much less sensitive than the surrounding skin that pressure made on one is felt rather around than beneath the growth.

Prognosis.—Warts are mere disfigurements, which exert no influence of any kind on the general economy, and are not even productive of any local discomfort. As has been already mentioned, they will often disappear spontaneously, and this event is far more probable in the case of a child than of an adult; but they will sometimes resist cauterization and other severe measures with singular obstinacy, reappearing time after time in their former position shortly after they have been (apparently) destroyed.

Diagnosis.—Verruca may be mistaken for Molluscum or Spilus.

It may however readily be distinguished from *Molluscum contagiosum*, by the latter being lobulated and marked with a small depression, from which a milky fluid can be expressed; by its having a smooth, polished. and far more sensitive surface, and its being found on section to consist of a thin envelope inclosing a brain-like substance, as well as by its tendency to terminate either by ulceration or sloughing. In *M. non-contagiosum*, if the tubercle be plump, it will present on its smooth, soft surface a hilus, from which a tenacious wax-like matter can be squeezed. If the waxy contents and the hilus be absent, the tumour will be pendulous and flaccid. *Spilus* is always a congenital affection, the elevated spot is soft, is always darker than the surrounding skin, and is often surmounted by a tuft of hard hairs.

Treatment.—This consists either in removing the tubercles by ligature or excision, or in destroying them by means of various caustics.

If the ligature be employed, the little tumour should be pulled forward by means of a hook or forceps, and a thread of silk or a piece of thin silver wire be tightly applied round its base, so as to embrace, if possible, a small portion of the skin; or if excision be practised, the neck of the tumour should be put as before on the stretch, and a small portion of the neighbouring skin removed with it. A pair of scissors curved on the flat is the most convenient implement. If cauterization be preferred, the repeated application every other day to the surface of the wart of a glass brush dipped in carbolic, in fuming nitric, hydrochloric, or sulphuric acid, or in a strong solution of chromic or of acetic acid, is the plan to be adopted. It may be necessary to state, that treatment by "erasion" (with a sharp spoon) is practically inapplicable to warts.

ACNE.

Synonyms.—The Stonepock; Pimples; Maggot pimples; Grog blossoms; Dartre pustuleuse; Bourgeons; Boutons; Couperose: Finnen; Liebesblümchen; Kupfergesicht; Varus; Gutta rosea: Comedones.

Definition.—The term Acne (*ἀκμή*, the prime of life, or *α* priv. *κνάω*, to itch) comprehends a variety of cutaneous affections, of which the principal phenomenon is either an excess or an alteration in character of the sebaceous secretion of the skin, or an inflamed condition of its sebaceous follicles.

Description.—These affections, although agreeing thus far with one another, yet differ so widely in degree and in appearance that they can scarcely be comprised under one general description. They will therefore be considered separately. They may however be conveniently described under two headings; the one including such as are due to an inflamed condition of the sebaceous follicles, and the other such as result from mere excess or alteration in quality of the sebaceous secretion.

Varieties caused by inflammation of the sebaceous follicles:—

Acne simplex begins by little, slightly, inflamed, elevated, red points. At the centre of each a small pustule gradually appears of the size of a pin's-head, surrounded by a red areola. The areola is not raised. The development of these little pustules is not accompanied by itching or any constitutional disturbance. After the pustule has burst, it leaves a little yellowish crust, which falls in a day or two, leaving a red stain, which slowly disappears. Sometimes the pustule is larger than above described, and its base is somewhat elevated; in such cases it is apt to leave behind it a minute cicatrix.

An eruption of Acne simplex is generally accompanied with a greasy, oily condition of the neighbouring skin, and is often associated with A. punctata. It is kept up by a succession of pustules, each of which lasts for about four or five days. It is met with on the face, affecting usually the forehead and temples, on the back between the shoulders, and on the upper part of the chest. It occurs generally in young persons, and has hence been termed Acne juvenilis.

Acne indurata begins by an indurated elevation of a violet-red colour, the summit of which soon suppurates. The pustule so formed lasts for several days. The indurated base on which it rests remains for some time after the disappearance of the pustule, and when it has subsided leaves behind it a small cicatrix. Besides these pustules, a number of violet-coloured tubercles appear, which in the course of a week or two may acquire the size of a pea, or even of a nut. As they enlarge they soften, and on manipulation it may be felt that their contents are fluid. These little abscesses may either point and discharge a thick curdy pus, or their contents may be reabsorbed. In either case they leave behind them small cicatrices. With this variety are frequently associated A. simplex and A. punctata.

A. indurata is seen more commonly on the face (viz., on the cheeks), and on the back, less commonly on the chest.

Acne rosacea (A. erythematosa) occurs only on the face. It is characterised by chronic erythematous redness, which occurs in irregularly-shaped patches, giving the skin a mottled appearance. The colour of the patches is in some cases a bright red, in others purplish. This condition is often accompanied with a varicose state of the venous radicles of the skin. In long standing cases the skin sometimes becomes roughened and rugous and covered with a furfuraceous desquamation. With this variety of Acne A. simplex is often associated, and occasionally other varieties of Acne. Its course is extremely chronic, and it lasts often for many years. It sometimes occasions a feeling of being flushed, and may even be accompanied by considerable tingling.

It is often limited to the nose, but it may affect the forehead, the chin, or the cheeks, or may be extended over the whole of the face. It is confined almost exclusively to persons in middle or advanced life, and is commoner with females than with males. It is temporarily aggravated by mental emotion, by the ingestion of stimulants, by exposure of the face to the sun, to the heat of a fire, or to a sharp wind. It is increased too by indigestion, constipation, and in females by the condition of system that precedes a menstrual period; in fact by everything that tends to congest the face.

In some cases the chronically-congested state of the skin of the nose leads to an irregular hypertrophy of it (*A. hypertrophica*). It becomes studded with red- or violet-coloured tubercles of various sizes, which may be either discrete or confluent—may be spread equably over the whole of the surface of the nose so as to enlarge it, sometimes to double its natural size without interfering materially with its shape; or may be developed only on particular parts of it so as completely to transform it.

Varieties caused by an excess of the sebaceous secretion:—

Acne punctata (varus comedo) appears as small black spots imbedded in the skin. The affected surface looks as if a number of grains of gunpowder had been shot into it. If the skin on either side of one of the spots be firmly[1] compressed, a consistent filiform white or yellow substance with a black head to it comes out. This is popularly called a skin-maggot. It consists of the inspissated contents of the dilated sebaceous follicle. Its black head is the result of the action of the atmosphere on the part which the gaping follicle leaves in contact with the air. The larger of these black spots is often surrounded by a small projecting ring of skin—the margin of the orifice of the dilated sebaceous follicle.

Simon (of Berlin) has discovered in these so-called "maggots" an actual epizoon, the *steatozoon folliculorum*. This parasite may be made apparent under the microscope by diluting the sebaceous matter with warm olive-oil.

This variety of Acne is often accompanied with a shining, greasy condition of the neighbouring skin. It is seen usually on the face, especially on the forehead and on the alæ of the nose. It is common too on the pinna of the ear, on the back of the shoulders, and on the upper part of the chest. It affects young persons. Its course is generally a chronic one. It may terminate spontaneously, either by the gradual escape of the sebaceous matter from the follicle, which slowly recovers its healthy tone, or by inflammation and suppuration of the follicle and the discharge of the sebaceous "core" in the midst of a small collection of pus.

Acne oleosa.—In this variety the sebaceous matter escapes freely from the follicles in the shape of a shining oily liquid which forms a greasy transparent film over the affected surface. The skin is somewhat redder than natural and has a sodden appearance, and the orifices of the sebaceous follicles are considerably enlarged. This condition of skin is met with usually on some portion of the face, generally the nose or on the scalp, but it occurs also on other parts of the body. When it affects the scalp, the hairs are bathed in a tenacious greasy substance, which towards their roots is of an oily consistence, but gets gradually more and more tenacious towards their tips so as to mat them together. This variety of Acne is often associated with A. punctata, A. indurata, or A. simplex. It affects young persons, and follows a chronic course.

Acne cerea.—In this variety the sebaceous exudation, instead of forming an oily film on the surface, concretes into a solid crust, which at first is soft, light-coloured, and easily detached; but afterwards gets harder, darker-coloured, and more adherent. On detaching the crust with the finger-nail, its under surface may sometimes be seen to be studded with little, short, white filiform projections, which are the contents of the sebaceous follicles which have been removed with it. It may be observed that it breaks with a waxy fracture, and that it is readily kneadable into various shapes—a character which at once distinguishes it from other cutaneous exudations. The surface laid bare by its removal is somewhat reddened, and the orifices of the sebaceous follicles are enlarged and gaping. The skin has sometimes too a sodden and unctuous appearance. It soon covers itself with a fresh crust. When the crust is hard and has lasted for some time, the subjacent skin has often a somewhat cicatricial appearance. This is generally associated with some other variety of Acne. It is situated generally on the face, but is not uncommon on the scalp, where it occurs as thin, dirty, grey crusts, which are apt to occasion more or less baldness. It is met with at all ages. It follows a chronic course.

Acne cornea is rarer than the preceding. It appears in the form of yellow, grey, or dark-brown conical or cylindrical projections, which are of a horny consistence. By compressing the skin at their base, they may often be expelled, so that it may be clearly seen that they are rooted in the orifices of dilated sebaceous follicles. This condition the author has seen associated with Ichthyosis cornea. It occurs on all parts of the body.

Causes. — *Acne simplex* is most commonly met with in young persons of either sex at about the age of puberty. *A. indurata* is also common with young persons, and is rare after the age of thirty. It has been ascribed to onanism; but the robust, healthy, and vigorous appearance of the majority of those who are affected by it is by no means in accordance with such a supposition. This variety as well as the *oily*, *waxy*, and *horny* varieties of Acne, may with greater propriety be referred to the influence of temperament as being affections that are almost constantly associated with the lymphatic temperament. In persons of this constitution the skin is comparatively thick, pale, opaque, sluggish, and greasy; in fact, exhibits in a moderate degree many of the characteristics that it presents in persons affected with these varieties of Acne. *A. rosacea,* which occurs usually in people of sanguine temperament, is an affection of middle age. It is occasioned and kept up by the various causes (already detailed) by which repeated flushing of the face is induced. It is commoner with females than males.

Duration.—Acne is a chronic affection, lasting usually for months or years.

Diagnosis.—*Acne simplex* may be mistaken for Ecthyma or Impetigo, but the pustules of Ecthyma are much larger and flatter and are surrounded with a broader areola. In Impetigo the pustules are confluent, and the raw surface left by their rupture exudes a plastic sero-purulent liquid, which concretes and forms thick crusts.

A. indurata is apt to be mistaken for Syphilis, more especially for "syphilitic Acne" and for "syphilitic Lichen;" but the pustules of the syphilitic affection may be distinguished from those of Acne by their situation, which embraces the limbs as well as the face and trunk; by the coppery colour assumed by their bases as they fade, and the co-existence of other symptoms of constitutional Syphilis. The small tubercles of the papular syphilide are to be known from those of Acne by similar tests as well as by the nature of their contents.

A. rosacea when it is attended with desquamation may be confounded with Eczema or with Pityriasis, but in Eczema itching is a more prominent symptom; there is more desquamation and the surface is moister, the eruption moreover is rarely confined to the face. In Pityriasis of the face the affected skin has a tawny-red rather than a purplish-red hue, and there is no enlargement of the venous radicles of the skin; the desquamation too is more abundant.

A. tuberculata may resemble "syphilitic Lupus," or scrofulous Lupus, but the tubercles of the syphilide are more translucent, their surface smoother, and the orifices of the sebaceous follicles are not enlarged. The diseased skin has a tendency to ulcerate and become covered with a thick crust, on detaching which a ragged deep ulcer is laid bare. The scrofulous tubercles may also readily be distinguished from those of A. tuberculata by their tendency to ulcerate and become covered with a crust which conceals an ulcer.

The secretion of *A. oleosa* is to be distinguished from ordinary sweat by its shining surface and oily consistence.

The crusts of *A. cerea* may be distinguished from those of Impetigo by their greasy nature and their malleability.

Treatment.—In Acne simplex, when the pustules are surrounded by deep red, somewhat elevated areolæ, and the patient is of sanguine temperament, the administration of saline laxatives will be requisite; the diet should be moderate in quantity and of an unstimulating kind; vapour-baths or the vapour-douche should be employed in the first instance and afterwards weak spirituous lotions. When the inflammatory character of the rash has subsided, a dilute solution of corrosive sublimate (gr. j or ij, ad ℥j.) applied tepid will complete the treatment. When the eruption occurs in females at the age of puberty in whom the menstrual function is imperfectly established, aloetic purgatives and warm hip-baths will be proper.

In Acne indurata, if the pustules be small, the frequent application of the tepid sublimate solution will sometimes suffice; but in the majority of cases more stimulating applications will be required, such as sulphur ointment (ʒj to ℥ss of sulphur to ℥j of simple ointment); ointment made with precipitated sulphur is preferable to that made with sublimed sulphur. Ointment of the iodide of sulphur (a favourite remedy) may be used, if preferred. If the pustules be large and interspersed with livid suppurating tubercles more decidedly stimulating applications will be called for. One of the most convenient is an ointment of the red iodide of mercury (gr. v., xxx. to ℥j); the application of this should be continued until the surface has become moderately inflamed, and in a few days' time, when the inflammation has subsided and the effect of the ointment can be judged of, its use should be resumed if necessary, and so on until the eruption has completely disappeared. In such cases recovery will be greatly hastened by opening the larger of the tubercles with the point of a lancet and expressing their curdy contents. An ointment of chrysophanic acid containing twenty per cent of the acid is a valuable remedy in Acne indurata, special care being of course taken to keep the ointment away from the eyes.

Most cases of this variety of Acne are associated with an habitually constipated condition of the bowels; when this is the case moderate but regular purgation materially aids the local remedies in subduing the eruption.

In many cases indurated Acne is associated with manifest indications of a scrofulous constitution; such cases are benefited by moderate doses of cod-liver oil with some chalybeate.

In Acne rosacea attention must be directed chiefly to the internal disorder on which the eruption depends. If there be any disturbance of the uterine function this will require to be corrected. When, as usually happens, the digestion is disordered, it will be necessary to rectify it; tonics should be given, the diet should be selected, and the bowels regularly acted upon if necessary. If the patient be of sedentary habits, regular and active exercise should be enjoined. In some cases, even amongst ladies of good position, habitual intemperance is the cause of the eruption. In such cases very little progress can be made unless the habits of the patient can be controlled.

The local treatment of Acne rosacea should consist in the application of astringents and moderately stimulating substances, such as tepid eau-de-cologne and water, camphor-cerate or weak tepid lotions of corrosive sublimate, of the acetate of lead, or of tannin. When there is much thickening of the skin stronger remedies are demanded, such as those already mentioned as suited to the indurated variety.

In Acne hypertrophica, when the eruption appears to result from free living, the diet should be moderate and simple. Saline purgatives with steel and aromatics should be prescribed and regular exercise be enjoined. Any of the tubercles that appear to be suppurating should be pricked with a lancet, and any distended and gaping sebaceous follicles should have their contents squeezed out. When the hypertrophy of the skin is not very considerable, moderately stimulating applications, such as sulphur ointment, will suffice, but when the skin is greatly thickened, such stimulation as for example is effected by a red iodide of mercury ointment is requisite to anything like speedy recovery.

In Acne punctata, when the gaping follicles are not very numerous, their contents should be squeezed out and some stimulant or astringent application be made to them, such as a strong solution of sulphate of zinc or of alum, or a little undiluted eau-de-cologne; but when the punctate eruption is copious other means must be had recourse to for unloading the follicles of their accumulated secretion. Bathing the affected surface with aromatic vinegar or with a dilute solution of potash or with benzine, favours the expulsion of the sebaceous matter. Gentle friction of the surface with a smooth piece of pumice-stone does so too. The vapour-bath and the vapour-douche have the effect of softening the inspissated secretion. But however much the *contents* of the follicles may be acted on, whether by chemical or mechanical means, the over-distended follicles, having lost all contractile power, are unequal to the task of expelling their secretion. The only effectual way of compressing them when they are at all numerous is by exciting such a degree of inflammation in the skin as shall produce palpable swelling, and then the distended substance of the skin, exerting an equally diffused and sustained pressure on the overgorged follicles, will completely empty them of their contents. For this purpose various stimulants may be employed, moderately strong creosote ointment, a mixture of croton with olive-oil, cantharides ointment, &c. When the follicles have been thus unloaded they should be encouraged to contract to their normal calibre by the daily application of some mildly stimulating or astringent substance. At the same time general measures adapted to give tonicity to the skin should be adopted, such as chalybeates, occasional laxatives, and the regular use of the cold bath.

In Acne oleosa the use of astringent lotions has been recommended, but the persistently greasy condition of the surface prevents them from coming into actual contact with the skin. It is therefore necessary to wash off the oily film by means of benzine each time before they are applied. Solutions either of tannin alum, sulphate of zinc, or acetate of lead, may be used, but mixtures of creozote with oil, or of a small proportion of hepar sulphuris with soft-soap, are preferable applications.

In Acne cerea the crusts should first be washed off by means of soft-soap, and then sulphur ointment should be used.

MOLLUSCUM.

Synonyms.—Elevures folliculeuses; Mycosis fungoides; Acne molluscum; Lupra; Acne varioliformis; Ecdermoptis.

Definition.—Molluscum (Mollusca, a nut with a soft shell) is a chronic disease of the skin characterised by more or less numerous sebaceous tumours varying from the size of a hemp-seed to that of a hazel-nut, rarely larger, either prominent or flattened, sessile or pedunculated, usually of the colour of the skin, but sometimes tinged with pink or with brown, marked generally with a hilus, and containing atheromatous matter which may be squeezed out through the hilus. There are two varieties of the disease, the non-contagious and the contagious.

Causes.—(Of M. contagiosum), childhood, contagion; (of M. non-contagiosum), the wearing flannel next the skin, neglect of ablution, middle or advanced life.

Description.—*M. non-contagiosum* appears on various parts of the surface in the form of minute indolent tumours, which augment very slowly in size. These are usually somewhat constricted at the base; the neck either may be thick and short, so that the little tumour is sessile, or may be narrow and long, so as to make it pedunculated and pear-shaped. (M. pendulum): some of the tubercles are rounded, plump, and of a pretty firm consistence, others are withered, flattened, and flabby. The surface has usually all the appearance of healthy skin, sometimes however it is slightly tinged with brown. In those that are plump the skin appears a little stretched, in those that are flabby it is thrown into folds, and has a relaxed and wrinkled appearance. There is usually to be found at some part of the tumour, generally near the centre, but sometimes on one side, a small cœcal depression. This is more obvious in those that are plump than in those that are flaccid; in some of the former it is not cœcal, but, though plugged up by a small comedo, communicates freely with the interior of the tubercle, and on compressing the little tumour a long, thin, white worm of inspissated sebaceous matter may be forced out through it. On section of one of the plump tubercles it is seen to consist of a fibro-cellular envelope containing an atheromatous substance. On incising a flabby one it is found to be merely a small pouch of redundant skin inclosing a little cellular tissue. The plump and sessile tumours are an early stage of the pedunculated and flabby ones, and are hypertrophied sebaceous follicles. There may be only one or two, or there may be a great many; they do not give rise to any sensation, neither are they at all tender to pressure, nor do they in any way affect the general health. They are more common on the trunk than elsewhere, and in the middle-aged and elderly than in young persons or children. They have no tendency to terminate spontaneously.

M. contagiosum, although resembling the preceding variety in general appearance, yet differs from it in many important particulars. Thus it is commoner in infants and young persons than in the aged; it is, as its name expresses, contagious; it is commoner on the head, neck, and limbs than on the trunk; the tubercles are of quicker growth and their contents are of a different character. The little tumours, which are hard, globular, and sessile, having a constricted base, vary in size from that of a large pin's-head to that of a pea or more; their surface is smooth and shining; their colour is either the natural colour of the skin or slightly redder, and they are usually semi-transparent. They present in the centre or at one side a small grey or black depression, from which five or six linear indentations radiate, dividing the mass into imperfect lobules, and giving it the appearance of a tomato or a rock-melon. On compressing the tumour laterally a small quantity of a milky fluid may be made to spurt out from the depression on its surface, but the bulk of its contents remains within unless the tumour be burst across by the pressure; if this happens a lobulated, glistening, white brain-like mass of nearly the size of the tumour itself is enucleated, and the thin membranous envelope is all that remains attached to the skin. The tubercles are of slow growth, taking several months to attain the size of a pea; they are developed without pain or itching; after attaining a variable size they may terminate spontaneously; the little tumour either is destroyed by suppuration, or becoming strangulated as it increases in size, sloughs off; whichever happens a small sore is left which soon cicatrises. The affection may consist of only a very few scattered tubercles confined to a limited region, or it may be constituted by a great many of them extensively spread and thickly strewn. The tubercles are not developed all together or even in crops, but follow one another in irregular succession, so that at any given time different tubercles will be seen in different stages. Their favourite situation is the face, especially the forehead, but they appear too on the scalp, the breasts, the limbs, and the genitals, and less commonly on the trunk itself. They are distended sebaceous follicles. Their contagiousness has been denied by some authors of repute, and held in doubt by others, the author has however met with cases where he has been enabled to trace the introduction of the disease into families, and its spread through them, so clearly to contact with affected persons, as to allow of no doubt, at least in his own mind, of its contagious character.

Prognosis.—By neither of the varieties of Molluscum is the general health in any way affected. In the non-contagious variety the tubercles, having attained a certain development, become stationary and continue indefinitely. In the contagious kind, however, they may terminate spontaneously either by suppurative inflammation or by gangrene. In either kind under suitable treatment speedy recovery may be obtained.

Diagnosis.—The constricted base, the indolent character, and the chronic course of these little tumours; the nature of their contents, and the presence of a hilus on their surface, suffice to distinguish them from any other elevations on the skin.

Treatment.—In M. non-contagiosum if the tubercles are plump, firm, and of but small size, their sebaceous contents should be pressed out, the orifice having been enlarged if necessary with a lancet; after this the ung. hyd. iod. rub. should be applied once or twice so as to cause inflammation of the enlarged follicle. In other cases the little excrescences should be snipped off with a pair of curved scissors.

If M. contagiosum has appeared in a family or a school, care should be taken to isolate the children that are affected from the rest, until the disease has disappeared. When the tumours are few, each of them should be incised with a lancet, and their contents enucleated by gentle pressure, the spots should then be touched with nitrate of silver. When from the number of the tubercles this mode of treating them is inconvenient, the application of some ointment that is at once stimulating and calculated to destroy a vegetable parasite should be had recourse to; the unguentum iodi compositum, or an ointment of the green iodide of mercury (gr. x. ad. ℥j) are suitable applications for the purpose.

SECTION VIII.—MACULÆ.

The Macular diseases of the skin are Nævus, Lentigo, Ephelis, and Vitiligo.

NÆVUS.

Synonyms.—Mother's Mark; Envie; Muttermaal; Maculæ maternæ; Ligna; Nævus sigillum.

Definition.—A Nævus, in the sense in which the term is understood here, may be defined as a congenital permanent discoloration of the skin, in some cases with, in others without, elevation beyond the surface of the normal skin.

Causes.—Nothing is known as to the determining causes of these malformations. They are popularly supposed to be occasioned by the longings of the mother during the time of her pregnancy for various delicacies which are thought to bear some sort of resemblance to the stains, *e.g.*, for claret, port-wine, or slices of ham; or to result from her having been startled by some object of aversion which resembles the mark, *e.g.*, a toad or a mouse.

Description.—Nævi are of two kinds, the pigmentary and the vascular.

The *pigmentary Nævi*, or Moles, (or Spili,[1]) occur usually as small rounded stains, which may vary in colour from a faint tawny yellow to a dark brown. The cutis is sometimes raised and thickened, and the discoloured spot is often surmounted by a tuft of hairs. In some cases the Spilus, in place of being small and rounded, is extensive and of irregular shape. The discoloration of the skin is due to an excessive (local) development of the pigmentary matter of the cuticle. When the mole is raised the elevation is due to simple hypertrophy of the cutis. The hairless moles are popularly known as toad-marks. Those that are surmounted by hairs are called mouse-marks or mole-marks.

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[1] σπίλος, a stain.

There may be very few of them, or they may be very numerous. The situations in which they most commonly present themselves the face, the neck, and the hands.

In some rare cases the greater part of the body may be cove with an extensive hairy mole, so that the individual, when stripp looks more like a monkey than a man.

Vascular Nævi appear either in the form of stains, which are le with the surface of the skin, or as tumours, which project beyond In the one case they are merely cutaneous, in the other they are m deeply rooted, and do not always involve the skin. The former ki results from an excessive development of the capillaries of the sk while the latter may be due to a congeries of dilated arteries, capillari or veins.

It is the former kind only—the port-wine stain, as it is popula called—that will be considered here.

The "port-wine mark" occurs usually in the form of purpl stains. The tint of these, however, in different cases varies considerab thus they may approach a brick-red, a claret-red, or a livid, blue colo Sometimes the stains are extremely small, presenting merely t appearance of a red dot with minute branching lines radiating fr it (Nævus araneus, or spider-mark), but often they are extensively spre When of large extent their shape is very irregular. They may occu any part of the surface, but are seen most commonly on the face a neck. They are quite superficial. Their colour, which disappears almo completely under the pressure of the finger, varies in intensity und the influence of such causes as augment or diminish the capilla circulation.

Diagnosis.—Both kinds of Nævus are such familiar and peculi objects that they can scarcely be confounded with any other lesion the skin.

Prognosis.—The pigmentary Nævi usually last for life without unde going any sensible change; in some cases, however, they gradual become less distinct. Vascular Nævi also remain in most cas without change during the whole of life. Some, indeed, soon aft birth become decidedly paler, while others in the course of a fe weeks altogether disappear; but those which continue unaltered f the first few months rarely undergo any subsequent change.

Treatment.—Both moles and port-wine-marks are considered by t majority of writers as incurable; that is to say, all the means whi they have employed with any success for the removal of the stai have always left a scar more disfiguring than the stain itself. If an thing be attempted, the cautious application of caustics, or if there much elevation of the stain and its area be very limited, the app cation of a ligature are the remedies suitable to moles.

Frictions with croton-oil or with tartar-emetic ointment, t application of blisters, the use of caustics, or (in an unvaccinat person) inoculation of the patch with vaccine-matter, are means attacking the port-wine marks.

A better method, however, of dealing with port-wine marks is to scarify the affected skin with numerous parallel linear incisions, arresting the bleeding by pressure and repeating the process when (after three or four days) the previous scarifications have healed; at each successive scarification the direction of the parallel incisions should be veered, so that they may decussate with the previous set of scarifications. Some ten or a dozen repetitions of the operation are requisite.

LENTIGO.

Synonyms.—Pannus lenticularis; Taches de rousseur; Lenticula. Freckles.

Definition.—A yellowish discoloration of the skin, the result of a lesion of the pigmentary secretion, congenital or acquired, occurring in the form of numerous minute spots, discrete or confluent, occupying the exposed parts of the surface.

Causes.—Congenital formation, youth, the lymphatic temperament, a fair complexion, exposure to the rays of the sun.

Description.—Lentigo occurs in the form of numerous minute, rounded, yellowish stains, the size of mustard- or lentil-seeds.

The tint of these varies a little; thus their colour may be a bright (saffron) yellow, a duller (tawny) yellow, a warmer (reddish) yellow, or a brownish yellow: in the last case the stains look like scales of bran sprinkled on the skin.

The spots may be either discrete and scattered pretty equably over the region occupied by them, or clustered and more or less confluent. They occupy most commonly the face and (to a less extent the) hands, but in persons of out-door occupation, whose chest and arms are habitually bare, *e.g.*, agricultural labourers or sailors, freckles are common on the forearms and upper part of the chest.

They are congenital, or appear during childhood.

They are found generally in persons of fair complexion and lymphatic temperament, but especially in those who have auburn or red hair and *very white* skins, so that they appear to be produced by an unequal distribution rather than an excessive development of the pigmentary matter of the skin.

Freckles are not accompanied by itching or any other sensation, nor is there either elevation or desquamation of the discoloured surface.

Spots which in no degree differ from congenital or infantile freckles are produced in certain persons on exposure to the sun's light, and appear only in summer. Those who are permanently freckled are especially liable to these ephemeral freckles, so that the freckling appears intensified during the summer.

Diagnosis.—Lentigo may be mistaken for Ephelis, pigmentary Nævus, Chloasma, Macular syphilide, or the stains left by syphilitic Lichen.

But in Ephelis the patches are much larger than in Lentigo.

Pigmentary Nævi are never so numerous as freckles; their shape is more irregular and they are of larger size.

From Chloasma, Macular syphilide, and the stains that succeed to syphilitic Lichen, Lentigo may be distinguished by the same signs as Ephelis.

Prognosis.—The congenital or infantile stains may last for life, but they often disappear during adolescence. Those which are caused by the rays of the sun generally disappear when their cause ceases to operate. This latter kind, although as regards appearance they are essentially lentigines, are in their nature more closely allied to Ephelis than to the congenital Lentigo.

Treatment.—For the congenital freckles nothing can be done, but for those which are caused by the rays of the sun the same measures are appropriate as for patches of Ephelis of similar origin.

EPHELIS.

Synonyms.—Pannus hepaticus; Taches hepatiques; Lebersflecke; Sommerslecke; Tawny blotches; Macula solaris.

Definition.—A yellowish-brown discoloration of the skin, the result of a lesion of the pigmentary secretion, occurring in moderately sized patches.

Causes (predisposing).—Adult age, the female sex, a delicate skin.

(Exciting).—Exposure to the sun, the menstrual period, pregnancy.

Description.—Ephelis occurs in the form of yellowish-brown rhubarb-coloured stains. These stains at their first appearance may be no larger than a threepenny-piece, but they usually extend in a variable degree so as often to acquire the size of a crown-piece. As they increase in area their margin, which at first is tolerably regular, becomes more or less uneven.

The disease consists simply in a change of the *colour* of the skin without any induration or thickening of its substance or any desquamation of the cuticle. It produces no appreciable disturbance of the general health, nor is it usually attended with any local sensation. In some cases, however, there is slight itching of the discoloured skin. The colour of the stains does not vary much, but they occasionally exhibit somewhat more of a warm yellow, or on the other hand more of a cold grey tinge, than is implied by the description "rhubarb-coloured."

The stains are not congenital and do not often appear before adult age has been attained; they occur usually on some part of the patient's fore-surface, but are far commoner on the face than in any other situation. In females they frequently appear soon after the commencement of pregnancy, disappearing shortly after delivery. In such cases the stains usually affect the forehead chiefly, and to a less extent the cheeks and upper lip. In some women they appear at the menstrual periods, disappearing during the intervals.

The occurrence of Ephelis may often be traced, however, in either sex to prolonged exposure to the rays of the sun in hot weather. When thus produced, the disease commonly appears on the forehead, but amongst agricultural labourers who work with the chest and arms uncovered, it often affects the upper part of the breast, the forearms, and the hands. When due to solar influence, Ephelis will generally fade and often disappear altogether during the winter months.

Diagnosis.—Ephelis may be mistaken for pigmentary Nævus, for Chloasma, for Macular syphilide, and for the stains which succeed to syphilitic eruptions on various parts of the body, or which follow chronic Eczema of the legs. But pigmentary Nævus is always congenital.

In *Chloasma* there is branny desquamation of the cuticle, and the spores of the microsporon furfur may be detected by means of the microscope in the epithelial scurf; the eruption too is attended with considerable itching, and is situated generally on the front of the chest, whereas Ephelis is more common on the face.

Macular syphilide is rare; its stains are of a deeper colour than those of Ephelis, and are usually accompanied with other symptoms of Syphilis.

The tawny stains which are left by syphilitic eruptions or by Eczema of the legs, may be distinguished by their having more of a chocolate and less of a rhubarb tinge, and by the history of an eruption having preceded them.

Prognosis.—The stains sometimes disappear with the cause (uterine or solar) which has given rise to them, but they are often chronic, they exert no influence on the general health.

Treatment.—If there be any derangement of the uterine function this should be corrected. If, however, sunlight appears to be the cause of the discolorations, exposure to the sun should be avoided, or at all events a veil or shade of some sort should be worn.

As internal remedies, the various sulphurous mineral waters, the Vichy water, or potash water, and mild laxatives, have been prescribed.

As local remedies, benzoin fumigations, ointment of the sulphuret of mercury (gr. x. to ʒj); lotions of corrosive sublimate (gr. j to ℥j); of sulphuret of potassium (gr. xij to ℥j); of sulphate of zinc, or acetate of lead (gr. viij to ℥j), or of the two mixed together so as to form a solution of acetate of zinc; warm douches of sulphurous mineral-water; the emulsion of bitter-almonds; weak spirituous lotions; lotions of the dilute mineral acids or of liquor-potassæ (ʒss to ℥j), or of Goulard's extract (♏ x. to ℥j); chlorine-water; ointments of the subcarbonate of potash or of soda (ʒj to ℥j); Oxymel,—are the remedies most employed.

VITILIGO.

Synonyms.—Leucoderma; Achroma; Ephelis alba; Macula alba; Macula albida; Alphoderma; Leucopathia.

Definition.—A milky-white discoloration of the skin (the result a lesion of the pigmentary secretion), congenital or acquired, occurri in the form of rounded patches of variable size.

Causes.—Nothing at present is known of the causation of Vitilig

Description.—Vitiligo occurs in the form of more or less extensi rounded (oval or circular) patches of blanched skin; the patches co mence as small white dots, which very slowly increase in size. T disease consists simply in an alteration of the *colour* of the sk without any modification of its substance or alteration of its surfac the skin remains as thin, as supple, and as smooth as usual: but h over a limited area exchanged its natural colour for a dead-whi appearance. If there be hairs on the patches they will be white als The skin immediately surrounding each of these white patches is of dusky-brown hue, and in proportion as the white patches get large the skin around them becomes more deeply tinged with brown. S that the disease seems to consist rather in an uequal distribution tha in an absence of the natural pigmentary constituent of the cuticl The whiteness of the patches ceases abruptly, so that their margins whether rounded or sinuous, are always clearly defined, and they ar rendered still more conspicuous by the more than naturally dark ski that surrounds them. The dark areola which surrounds the whit patch, although it ceases abruptly at its inner margin, yet at its oute border fades insensibly into the natural colour of the skin. Th colouring matter is not always collected equally and uniformly aroun the blanched spaces; at one part of their circumference it may form black patch, while at another point the areola may be altogethe wanting. Vitiligo occurs on all parts of the body, but especially o the uncovered parts (face, neck, and hands), and on the hair parts of the body (for instance, the scalp, the axillæ, and th pubes). It is a comparatively rare disease. It is commoner amon the dark than among the fair races of mankind. In the negro it con stitutes what is known as the piebald negro. Its course is alway very slow and gradual.

Prognosis.—When congenital it is not likely ever to disappear when acquired it occasionally disappears spontaneously, after which i may sometimes gradually reappear.

Diagnosis.—Vitiligo is likely to be confounded with Addison disease, but in the latter there are no definite patches whiter than th natural colour of the skin. There are dusky-brown patches, but the are diffused and their margins are in all directions gradually lost i the natural colour of the unaffected skin. There are no concav well-defined margins to any part of the stains. The discoloration of th skin is, in Addison's disease, attended with marked deterioration the general health. In Vitiligo not so. Again, while the latter is th effect of an unequal distribution of the pigment of the cuticle witho alteration of its quantity, so that part of the skin is darker, pa lighter, than natural, the former is characterised by an excessi development of the pigment of the skin, so that parts of the surfac are darker but none lighter than is proper to the individual in health.

Treatment.—No remedies are known which are of any efficacy in the treatment of Vitiligo.

SECTION IX.—SYPHILIDA.

Synonyms.—Syphilides; Syphilitic eruptions; the Secondaries; the Venereal disease; the French disease; Le mal anglais; Le gros mal; Le mulet épidémique; La vérole; La grande vérole; La gomme; Die Lustseuche; Venerische krankheit; Lues venerea; Morbus fœdus; Morbus venereus; Lues syphilitica; Pudendagra.

Definition.—The term "Syphilide" comprises all diseases of the skin that are developed under the influence of syphilitic infection, excepting the primary syphilitic ulcer or chancre.

Causes.—The only causes are contagion and hereditary transmission.

General description.—Although differing considerably from one another not only in aspect, but in situation, course, and many other details, the Syphilides have nevertheless certain characters in common which distinguish them as a class from other diseases of the skin.

The *colour* of the Syphilides is peculiar; it has been compared by some to that of metallic copper, by others to that of the lean part of a ham; for this tint to be present it is not necessary that the surface should continue unbroken; thus it may not only be present in exanthematous, papular, and tubercular Syphilides, but may be seen also around the edges of syphilitic pustules, blebs, and ulcers.

The *shape* of the eruption is frequently annular; thus it may form complete circles or ellipses or these may be incomplete, so that a patch may assume a crescentic or a horse-shoe shape, or two complete rings may be in juxtaposition so that a figure of 8 be formed, or two incomplete rings may become fused so as to resemble the letter S or the letter E.

A Syphilide is rarely attended with itching or smarting.

The often *mixed character* of a syphilitic eruption—that is, the coexistence of several distinct varieties of Syphilide—is another means of recognising its nature; thus syphilitic rashes, scales, papules, and pustules may occur together on the same individual.

The secondary products, such as the scales, the crusts, the ulcers, and the scars of a Syphilide, have characters which distinguish them from those of a "simple" cutaneous disease. *The scales* of a squamous Syphilide are scantier, finer, smaller, and more adherent to the surface beneath than those of a simple squamous affection. *The crusts* left by a bullous or an ulcerating tubercular Syphilide are much thicker and harder and are more firmly adherent than the crusts of a simple eruption; they are usually of a dark green colour; their surface, which is raised at the centre, is often marked by a series of concentric rings showing a stratified arrangement of their substance, and may be dotted here and there with little nodular projections, so that the crusts may bear a pretty close resemblance to limpet-shells. *The ulcers* that are produced by a Syphilide are generally circular and have clean-cut perpendicular edges and grey pultaceous floors; their margins, as before mentioned, are of a coppery hue. *The cicatrices* left by the ulcers retain for some time the tawny-brown hue of the eruption that has preceded them; after this has faded they may often still be recognised by their preserving the traces of an annular or a crescentic arrangement.

No one however of the characters above detailed is pathognomonic of a Syphilide.

If, for example, the tawny hue of an eruption be regarded as an absolute proof of its syphilitic origin, simple Psoriasis would at once be brought under the category of the Syphilides; on the other hand, the absence of this hue connot be regarded as positive evidence of the simple nature of an eruption, since many of the Syphilides at their commencement do not at all exhibit it, but are of a bright rosy-red colour, assuming the tawny tint only as they are beginning to fade.

The annular arrangement too is by no means *peculiar* to eruptions of syphilitic origin, since it is found also in Lichen, Lupus, Psoriasis, and Herpes circinatus; nor is it even a constant symptom of syphilitic cutaneous disease—it is wanting in syphilitic Acne, syphilitic Lichen, and in mucous tubercles.

Again, itching and smarting are not always completely absent from a syphilitic eruption and are alike rare in (scrofulous) Lupus.

Nor is it an invariable rule that one variety of cutaneous Syphilis should be accompanied by some other; for instance, syphilitic Roseola often occurs singly; so do mucous tubercles.

In like manner exception may be taken to any one of the secondary products of cutaneous Syphilis, being considered as constituting by its presence or absence a pathognomonic or absolutely distinctive sign of the syphilitic or non-syphilitic nature of an eruption. But enough has been said to show that each of the phenomena enumerated above as generic features of the Syphilida may in its turn be either altogether wanting or but faintly apparent, and that there is no single characteristic of cutaneous Syphilis which taken separately may not be more or less simulated by non-syphilitic eruptions.

In determining whether a disease of the skin be syphilitic or not much assistance is to be derived from a knowledge of other changes produced by syphilis besides those wrought in the skin itself:—*redness of the fauces, opaline infiltration (in patches) of the mucous membrane* of the cheeks, fauces, posterior surface of the pharynx, uvula, tonsils, palate, tongue or gums; or ulcers in the same situations.

Chronic laryngitis.—Indicated by a husky whispering quality of the voice.

Mucous tubercles (often complicated with deep fissures) at the angles of the mouth, at the nostrils, the arms, and the vulva, or the prepuce and scrotum; or condylomata, or cauliflower-excrescences of anus, vulva, or prepuce.

Neuralgia affecting both sides of the head over the frontal and parietal regions.

Rheumatic or *osteocopic pains* (as they are termed) felt along the bones and in the joints of the limbs, becoming worse at night-time. *Thinning of the hair* of the scalp and face, sometimes in a very considerable degree.

Periosteal swellings, exostoses, caries of the bones of the cranium or face, *circumscribed indurations of the sub-cutaneous cellular tissue, syphilitic iritis,* the *syphilitic cachexia, &c.*

The various forms of syphilitic cutaneous disease may be arranged in eight groups as follows:—

Vegetative, exanthematous, vesicular, squamous, papular, pustular, bullous, and tubercular Syphilides.

Some of the Syphilides appear soon, some late after the occurrence of syphilitic infection. In other words, some may be spoken of as the earlier and some as the later manifestations of the syphilitic diathesis. To the former the name of "secondary," to the latter that of "tertiary Syphilides" has been given. It would be unwise to abolish these distinctions (although it must be admitted that sometimes the so-called "tertiary" appear soon, and the "secondary" late after syphilitic infection) provided that no more than a just significance be attached to them; that the term "secondary Syphilide" be held to mean only a "species of syphilitic eruption which is not usually associated with depreciation of the general health, does not occasion any notable destruction of the skin, and which when it occurs generally makes its appearance soon after the acquirement of the syphilitic diathesis," and that a converse meaning be attached to the term "tertiary Syphilide."

With this understanding it may be said in general terms that the vegetative, exanthematous, vesicular, squamous, and papular Syphilides belong to the "secondary," and that the pustular, bullous, and tubercular Syphilides compose the "tertiary" division.

Varieties of Syphilitic Cutaneous Disease.

Vegetative Syphilides.—These are among the commoner of the manifestations of constitutional Syphilis, and are developed often within a few weeks after syphilitic infection. But although they may fairly claim to be classed among the secondary symptoms of Syphilis, inasmuch as they may make their first appearance many weeks after infection, and in situations remote from the point of inoculation, they differ from all the other secondary symptoms of Syphilis in being endowed with the capacity of playing also the part of primary Syphilis; they share with the chancre the property of transmitting syphilitic infection.

The vegetative Syphilide is of two kinds, the flat and the prominent The *flat variety,* known under the name of "mucous tubercle," or "mucous patch," commences as a small rounded, slightly-elevated, rose-red patch, which soon becomes excoriated. The raw surface thus produced, which may be either of a bright pink or an ash-grey colour, is composed of numerous moist flattened granulations, which are marked out by very shallow linear depressions. It presents such an appearance as would be produced if an extremely fine net were drawn tightly over a slightly convex surface of moist mucous membrane, so that the pulpy substance of the mucous membrane might be forced to bulge a little through the meshes of the net. The appearance of the surface does not convey the impression of an open sore, the granulations, if they may be called so, are too regular in their arrangement, too flat and too tough (not bleeding when they are rubbed); they appear to be protected by a tolerably tough opalescent membrane, the surface of which has a moist glistening appearance. Altogether the term "mucous patch" is a very apt one.

The outline of the mucous patch is usually regularly circular or oval.

Its size ranges from that of a sixpenny-piece to that of a florin.

Its surface, which is generally slightly convex, may at its circumference be even with the level of the surrounding skin. But usually the edge of a fully-developed patch is raised a line or two above the level of the sound skin, so that the raised patch is abruptly limited, its margin being connected with the lower level of the sound skin by a wall of tense, thin, dusky-red skin. Sometimes the surface of the mucous patch is sprawled out, so that its everted margin overlaps the distended ring of skin which limits its root. The mucous patch then presents a really "vegetative" appearance; this is the form that it ordinarily assumes.

When the mucous patch occupies an exposed situation (*e.g.* the cheek) the liquid secreted from its surface dries up into a tolerably thick yellowish crust, on detaching which the mucous tubercule is disclosed exhibiting its special appearance. But when, as more commonly happens, a mucous patch is in a cleft (as it is when developed on the margin of the anus), so that its surface is in contact with an opposed surface of skin, no crust is formed, but an abundant viscid, fetid, irritating mucous discharge exudes from the patch, causing much redness and itching of the surrounding skin.

Mucous patches are almost invariably present in cases of infantile Syphilis. Amongst adults they are commoner with syphilitic women than with syphilitic men.

They occur in situations where the skin at the various orifices of the body joins mucous membrane; in the various mucous passages near to their external orifices; on the skin in the neighbourhood of the orifices of mucous passages; and in places where the skin is fine and habitually moist, as in folds where the opposite surfaces of the skin are frequently in contact.

Thus they are common on the vulva, at the margin of the anus, on the lips, on the margins of the nostrils, in the lower part of the rectum and vagina, on the tongue, on the lining membrane of the cheeks, on the tonsils, on the uvula, on the posterior wall of the pharynx, on the cheeks, in the clefts of the alæ of the nose, on the prepuce, in the cleft of the nates, on the scrotum, on the upper part of the inner surfaces of the thighs, in the groin, in the umbilicus, in the axillæ, between the toes and around the nails.

Mucous patches are rarely attacked by ulceration. When they are disappearing they gradually dwindle (are atrophied), their secretion is diminished, and they become the seat of a moist flaky desquamation, till at last nothing remains of them but a livid stain, which gradually fades, leaving no scar behind it.

The *prominent* kind of vegetative Syphilide is known under the names of "condyloma," "syphilitic wart," "cauliflower excrescence," &c.

This, like the mucous tubercle, presents a somewhat different aspect, accordingly as it is developed in an exposed situation or occupies some cleft, where it is protected and kept moist by the contiguous surfaces of the cleft.

In the one case it will be an ash-grey dry wart, hard and rasp-like to the touch; in the other it will be composed of semi-transparent, pink-coloured, moist, ill-smelling, fungous granulations. It may be sessile or pedunculated. Its constituent papillæ may be confluent, or, as is often the case, distinct, so that the growth is digitate.

It occupies the same situations as the mucous tubercle.

Diagnosis.—The mucous patch can scarcely be confounded with any other lesion of the skin. The diagnosis of the syphilitic cauliflower excrescence, however, is not always so clear, since growths which are undistinguishable in appearance from syphilitic warts are to be met with about the anus and genitals in cases where there is no reason to presume that they are due to syphilitic infection. In instances where the nature of the warty growths is a matter of doubt, the history of the case and the presence or absence of other syphilitic symptoms will furnish aids to the formation of a diagnosis.

Exanthematous Syphilide ("Syphilitic Roseola") is one of the most frequent and early of the symptoms of constitutional Syphilis. It appears in rounded patches of the size of a sixpenny-piece or a shilling, having irregular broken margins. Their colour, at first of a bright rosy hue, acquires afterwards a tawny coppery tint, and finally disappears as a cold-grey stain. Both at its commencement and at its termination, however, the eruption is very faint and may easily be overlooked. The patches, which are tolerably numerous, may be either distinct from or may run into one another; in the latter case a sort of irregular web is formed, which, enclosing islets of unchanged skin, gives a mottled appearance to the surface. When recent the eruption can be made to disappear momentarily under the pressure of the finger, but later on it loses this quality. Its appearance is preceded usually by slight febrile disturbance lasting a day or two.

The roseolous rash generally lasts for one or two months, affecting some portion of the trunk or upper part of the arms or thighs. It makes its appearance about a month or two after primary infection. It is scarcely ever accompanied either by itching or smarting; as it fades a transient desquamation will sometimes take place, but no ulceration or cicatrization. It leaves behind it, however, dull-grey stains, which often persist for a long while. It coincides occasionally with other of the earlier manifestations of secondary syphilis—redness or opaline infiltration of the mucous membrane of the mouth or fauces, mucous tubercles, rheumatic pains in the limbs, papular and other early Syphilides.

The Prognosis as regards the eruption itself is favourable since it is a mere rash, which disappears after a comparatively short duration, leads to no destruction of tissue, and leaves behind it no permanent trace; but viewed as a symptom of constitutional syphilitic infection its appearance indicates that the subject of it is more or less liable to further manifestations of constitutional Syphilis.

Diagnosis.—This will be facilitated by the co-existence of a chancre, or at least of the recent cicatrix of one on the genitals. The affection may, however, be mistaken for Measles, Scarlatina, or Roseola.

But in *Measles* the eruption commences on the face, has a crescentic arrangement, and is of a mulberry colour, the eyelids are much swollen, and there is coincident coryza and bronchitis. In *Scarlatina* the eruption is most intense on the upper part of the chest, is punctiform, and is of a bright scarlet colour, the redness of the throat, too, is of a brighter hue than the dusky redness of syphilitic angina. *Roseola* is attended with itching, its duration is much briefer, and it fades without undergoing the changes of colour peculiar to the exanthematous Syphilide.

Vesicular Syphilides are among the earlier manifestations of constitutional Syphilis; they are not, however, of frequent occurrence. They have been named respectively after the "*non*-specific" eruptions that most resemble them— '*Syphilitic* Eczema," "Herpes circinatus," "Varicella," and "Herpes phlyctænodes."

"*Syphilitic Eczema*" is characterized by the development of numerous small vesicles clustered together on a reddened surface, which soon acquires a tawny hue. The eruption occurs in irregularly-shaped patches of limited size; the vesicles last for several days, after which they either shrivel and are replaced by furfuraceous scales, or become ruptured and are succeeded by minute crusts, which on falling disclose small brown stains, which in their turn slowly disappear. The eruption may be prolonged over a variable period by a succession of several crops of vesicles.

Syphilitic Herpes circinatus appears in the form of small elevated reddened patches surmounted by clusters of minute vesicles; the circumference of each of these patches rapidly extends at the same time that the centre subsides and fades, so that the eruption speedily assumes the form of narrow reddened elevated rings, each enclosing an area of skin which is either of the natural colour or but slightly tinged with red: the rings are surmounted by minute vesicles, or more commonly by minute furfuraceous scales, and soon acquire a tawny hue. The patches may be either few in number and confined to a limited region or extremely numerous and widely spread; in the former case they occur usually on the sides and back of the neck, in the latter they are thickly scattered over the greater part of the body and give it a peculiar and variegated appearance.

Syphilitic Varicella.—In this variety the vesicles are large, distinct from one another, and scattered irregularly over the surface; they are surrounded by slightly elevated red areolæ, which steadily acquire a coppery tinge. After lasting for a week or ten days the vesicles burst and are replaced by dark-coloured, greenish-brown, adherent crusts, which at length separate, leaving in their place brown stains, which persist for some time. When the eruption is more or less clustered so that the areolæ of several neighbouring vesicles become fused, it has been termed "*Syphilitic Herpes phlyctænodes.*"

Vesicular Syphilides appear within a few months after contagion and last a month or two; they coincide usually with such other syphilitic symptoms as have been mentioned in the description of the exanthematous Syphilide.

Prognosis.—What has been said respecting "syphilitic Roseola" on this head applies equally to the vesicular Syphilides.

Diagnosis.—The comparatively long duration of the vesicles individually, taken with the general diagnostic signs of cutaneous Syphilis, will suffice usually for the recognition of syphilitic vesicular eruptions.

Squamous Syphilide, one of the earlier and commoner of the syphilitic eruptions, is of three kinds—"syphilitic psoriasis guttata," "syphilitic psoriasis circinata," and horny Syphilide.

"*Syphilitic Psoriasis guttata*" is characterised by the appearance of reddened disc-shaped elevations of the skin, which are scantily covered by small, dry, dingy-white, thin scales. The inflamed disc soon assumes a coppery tinge, and its circumference, which is bare of scales, forms a tawny-red areola around its scale-covered portion. The patches, which are as a rule rather oval than rounded, vary in size from that of a split-pea to that of a shilling, rarely exceeding the dimension of the latter; they are distinct from one another though sometimes closely set. This variety is usually spread pretty extensively over the whole surface of the body, but is perhaps more developed on the inner surfaces of the limbs than elsewhere.

"*Syphilitic Psoriasis circinata*" begins as slight, scurf-covered, reddened elevations, which as they extend subside at their centre so as to acquire the form of rings. These papular rings, which may be complete or incomplete, are of a tawny-red colour, and are surmounted by small, thin, dingy-white scales; their average diameter is that of an ordinary finger-ring. The disease may remain almost stationary in this condition for some time, till at length the scales disappear and the elevation subsides and is replaced by a deeper-coloured tawny-brown stain. This variety occurs on the face, neck, and limbs.

Horny Syphilide.—This affects exclusively the palms of the hands and the soles of the feet. It appears first as rounded, slightly elevated, tawny-red patches of the size of a split-pea; these gradually increase in size till they acquire the diameter of a fourpenny- or a sixpenny-piece. Gradually the epidermis covering them becomes thickened, dry, hard, opaque, and yellow—assumes in short a horny appearance, and at length begins to separate in flakes. The disc of altered epidermis is surrounded by a tawny-red areola. Sometimes a large patch of this kind will completely cover the posterior part of the sole of either foot, the epidermis acquiring in this situation an extraordinary thickness and separating in large thick plates. After a time the desquamating patches are replaced by yellowish-brown stains, which gradually disappear.

The Prognosis of the squamous is the same as that of the exanthematous Syphilides.

Diagnosis.—"Syphilitic" Psoriasis may be mistaken for "simple" Psoriasis. But in simple Psoriasis the knees and elbows are almost invariably attacked, the face rarely so; the outer surfaces of the limbs and the lower part of the back are the regions chiefly affected; the scales are abundant and imbricated and have a nacreous lustre; the surface on which they rest is harsh and wrinkled; it is redder and less tawny than in the squamous Syphilide. A narrow dingy-white circle commonly surrounds the areola bordering the syphilitic scaly patch. This appearance is caused by a ring of altered and partially-detached epidermis; it is less frequently present in simple psoriasis, and in the latter disease is thicker and presents a nacreous lustre, but it is not (as has been supposed) a pathognomonic sign of constitutional Syphilis, nor is it limited to squamous eruptions.

The horny Syphilide can scarcely be mistaken for any other eruption.

The symptoms which have been already mentioned as distinctive of the Syphilida generally will facilitate the diagnosis of squamous Syphilides from non-syphilitic eruptions.

Papular Syphilide is the most common of all the syphilitic eruptions. It appears usually within two or three months after the occurrence of primary Syphilis, and is accompanied by other of the earlier symptoms of constitutional Syphilis. It is occasionally preceded by slight febrile disturbance, which subsides as the eruption comes out. It may coincide with apparently the most perfect general health.

Syphilitic papules are small, solid, firm, shining elevations, which contain neither serum nor pus. Their colour is at first, and for some time, a bright red or rose colour, which can be made to disappear momentarily by the pressure of the finger. Afterwards, as they are beginning to fade, they gradually assume a tawny tint, which, as they slowly disappear, becomes more and more pronounced. After this change of hue the colour of the papules will no longer pass away under pressure.

Soon after a papule has attained its full size its top becomes covered with a small white scale.

There are two varieties of papular Syphilide, the conical and the flat.

In the *conical* variety (the so-called "*syphilitic Lichen*") the papules are small and cone-shaped. They are distinct from one another, not confluent as in simple Lichen, and are thickly scattered over a large extent of surface.

In the *flat* variety the papules are much larger and flatter, and are less numerous. Their average size is that of a threepenny-piece. In these papules the tawny hue becomes deeper than in the conical kind.

Not unfrequently both varieties are found together.

Papular Syphilide affects especially the upper half of the body, viz. the face, the neck, the upper limbs, the back, and the chest. The average duration of each papule is from three to six weeks, but as the eruption usually comes out in a succession of crops the disease may in this manner be prolonged over several months. The papules terminate in resolution, leaving behind them either dull yellow-brown stains, or in some cases small depressed cicatrices, which for a long time retain the tawny hue, but at length disappear completely.

The prognosis of the papular is the same as that of the exanthematous Syphilide.

Diagnosis.—The eruptions for which papular Syphilide is apt to be mistaken are Lichen, Prurigo, Erythema (papulatum), Acne (indurata), and tubercular Syphilide.

However, in *Lichen* the papules are not shining; they are closely agglomerated, and are much smaller than the syphilitic papules. In *Prurigo* the papules are paler and of smaller volume than in the papular Syphilide, and they are surmounted generally by a small black crust. In *Erythema papulatum*, on the other hand, the papules are more voluminous than in the disease under consideration, and change soon from a rose-red to a purple hue. Their duration is much briefer than that of the syphilitic papules. In *Acne indurata* the coexistence of other varieties of Acne, the fact that from some of the (suppurating) papules a curdy sebaceous matter may be expressed, the absence of the eruption from the chest, abdomen, and arms, and its somewhat purple hue, will suffice for distinction. But the diagnosis of papular Syphilide from either of the above-named diseases will be greatly facilitated by a recollection of the characters which have been already enumerated as distinctive of the Syphilida generally. From *tubercular Syphilide* the papular disease is distinguished by the smaller size of its pimples, by their not leading to ulceration, and by their not producing a permanent cicatrix. Again, syphilitic tubercles appear usually at a much later period after contagion than do the papules, and coincide with other of the later phenomena of constitutional Syphilis.

Pustular Syphilides.—At one time syphilitic eruptions were all of them believed to be of a pustular character; the term "pustular," however, can with propriety be applied only to three kinds of syphilitic eruption. These have received the names of Syphilitic Acne, Syphilitic Impetigo, and Syphilitic Ecthyma.

Syphilitic Acne, one of the earlier Syphilides, appears in the form of numerous isolated papules, each of which is surmounted by a small pustule. The average size of the pimples is that of a hemp-seed. The solid base, at first of a rosy-red colour, gradually acquires a brownish tint, and the small mattery head slowly dries up, producing a yellowish-brown crust, which at length falls off. The papule capped by it becomes absorbed, and is replaced by a small rounded depression of a tawny brown-red colour, which after a scanty desquamation either disappears completely or is replaced by a small rounded depressed cicatrix.

The pimples, which occupy usually a considerable extent of surface, may either be scattered equably over the region affected or be collected into groups, but are generally, in either case, perfectly distinct from one another.

The favourite situation of the eruption is the back, but it appears also on the face, upper limbs, and thighs.

Each pimple runs a chronic course, lasting for about a fortnight. The average duration of the disease altogether is about three or four months. The invasion of the disease is sometimes gradual, but in other cases an extensive surface becomes thickly covered in a comparatively short space of time. There is then slight febrile disturbance, lasting for two or three days.

This variety is often associated with conical papular Syphilide, in some places the papules predominating almost to the exclusion of pustules, in some being mixed in equal proportion with them, while in others the eruption will be exclusively pustular.

Prognosis.—The eruption rarely exceeds a duration of four months, exercises no apparent influence on the general health, except the febrile disturbance it sometimes occasions at its outset, and leaves but slight traces behind it.

Diagnosis.—It may be mistaken for Acne, but the Syphilide is generally more extensively spread. The grouped arrangement is one that rarely occurs in Acne. Acne is a more chronic disease, and it exhibits generally a purple tint. The scars left by Acne are linear and elevated, those left by the acniform Syphilide are rounded and depressed. In Acne there is more or less curdy matter contained in some of the pimples.

Syphilitic Impetigo is of two kinds, the superficial and the deep.

The *superficial variety* is one of the earlier syphilitic eruptions, and is associated often with syphilitic Roseola or with syphilitic Lichen. It appears in the form of small pustules. These are generally arranged in small clusters on rosy-red patches of skin, which soon acquire a tawny hue. The pustules are soon replaced by uneven, brown, somewhat thin crusts, which, falling, leave superficial cicatrices, which retain for some time a tawny-brown colour. The eruption is sometimes preceded for a day or two by slight febrile disturbance. It may be kept up for some time by a succession of crops of pustules. It is common on the hairy scalp, but it occurs also on other parts of the body.

The *deep variety* is a tertiary Syphilide, and occurs usually in cachectic persons, more particularly in those in whom the 'syphilitic cachexia' has become developed. It is a more chronic disease than the superficial variety. It begins by an elevated reddened patch, on which numerous confluent pustules appear. These become speedily replaced by a dark-green, uneven, tolerably thick crust, which conceals a moderately deep ulcer, having a coppery margin, clean-cut edges, and a greyish floor. After lasting for some time the crust falls, leaving depressed, well-marked, livid cicatrices, which gradually acquire a brown hue and at length become blanched. This variety is common on the face.

Prognosis—of the superficial variety as of syphilitic Acne; of the deep variety as of syphilitic Ecthyma.

Diagnosis.—Syphilitic Impetigo may be mistaken for simple Impetigo, but the latter occupies generally a more extensive surface, is characterised by a more abundant purulent secretion, which produces crusts of a lighter colour. The pustules have a briefer individual duration, the inflamed skin does not acquire a coppery hue, no ulcers are produced, nor does the eruption after it has disappeared leave behind it any cicatrices.

Syphilitic Ecthyma is characterised by the development of large isolated pustules, which become replaced by thick dark-coloured crusts covering ulcers.

There are two varieties of it, the superficial and the deep.

The *superficial* appears earlier and is less severe than the deeper variety. It commences in the shape of small, scattered, reddened patches, on each of which a pustule is shortly formed. The pustules when fully developed vary in size from that of a split-pea to that of a split-bean; they are prominent and rounded; their base is not solid and indurated as in the acniform syphilide, but they are formed entirely of a collection of pus; they are surrounded by a tawny-red areola, and after a short time they burst and the tenacious liquid they contain concretes into a moderately thick, rugged, greenish-brown, not very adherent crust, covering a shallow but characteristic ulcer: this at length heals under the crust, leaving a dusky superficial but often permanent scar, which becomes gradually blanched. This variety occurs on the scalp, where it gives rise to permanent circumscribed baldness; on the limbs, more especially the lower limbs; and on the nates. It is a not uncommon form of infantile Syphilis appearing a month or two after birth. But in infants I have noticed that it presents special characters which it may be well to mention. Thus it appears at first in the form of conical tubercles, which gradually suppurate throughout their whole extent, commencing at the summit, and it is peculiar too i affecting the palms of the hands and the soles of the feet.

The *deep* variety (which ranks among the tertiary Syphilides) commences as an elevated livid spot, which is soon replaced by a pustul containing sanious pus, and resting on a livid, slightly elevated, indurated base, which is broader than the pustule; the pustule give place to a thick nodulated crust elevated at the centre, of a dark greenish colour, let into the thickness of the skin and surrounded by a tawny-brown swollen areola. The crust conceals a deep ulcer with perpendicular edges, and a grey slough-like floor. After a time thi heals, and the crust falls, disclosing a well-marked, depressed, livid cicatrix, which gradually becomes bleached.

Syphilitic Ecthyma occurs more especially in the cachectic, whether the cachexia be more immediately due to the syphilitic virus o to some other cause. Thus it occurs in those who, contracting constitutional Syphilis, have had their vigour impaired by intemperance, debauchery, privation, or exposure; it occurs too in infants and in persons in whom the syphilitic cachexia has begun to manifest itself.

The superficial variety appears from six months to a year or mor after primary Syphilis, the deep variety usually much later. Th disease is generally, by a succession of pustular crops, prolonged ove a considerable period.

Prognosis.—This as regards the probable duration of the eruption and the extent of disfigurement likely to be occasioned by it, may b stated as being intermediate between that of syphilitic Acne and tha of syphilitic Rupia. The prognosis of the deep will be graver tha that of the superficial variety.

Diagnosis.—Syphilitic Ecthyma is very apt to be confounded wit simple Ecthyma (cachecticum), and the diagnosis is sometime difficult, unless the history of the case, and the presence or absenc of other indications of constitutional Syphilis be taken into th account. However, in the simple eruption the areolæ surrounding th scabs are purplish rather than coppery, the pustules in the majorit of cases are confined to the lower limbs, and the individuals attacke are generally either infants or aged persons. In the latter the diseas is often complicated with the characteristic indications of Prurig senilis, which disease the Ecthyma is in such cases secondary t syphilitic Ecthyma, on the other hand, occurs on all parts of th surface, including even the head and face, and is commoner in adu age.

The Bullous Syphilide (*Syphilitic Rupia*) is one of the latest a most inveterate of the Syphilides. It appears in the form of tolerab large blebs; these are of an irregularly rounded outline, have a f and somewhat wrinkled surface, project but slightly above the lev of the skin, and contain a turbid sanguineous serum, which is of brownish colour. They are surrounded by a red areola, whi speedily assumes a tawny-brown tint. After lasting for a short tin the bleb becomes ruptured, and the liquid it contains dries up into a thick, hard, very adherent, dark-brown, or more often dark-green crust, beneath which is an ulcer. The firm adhesion of the scab, the extremely chronic character of the sore which produces it, and the tendency of the latter gradually to spread, produce a peculiarity in the shape of the incrustations which is very characteristic of the disease; thus they are generally moulded in the shape of an oyster-shell or of a limpet-shell. This arises from their being produced in successive layers, which, as the ulcer spreads, become progressively larger and larger. When the exudation is scanty, and the spread of the ulcer comparatively rapid, the oyster-shell shape results; when the purulent secretion is profuse, and the ulcer extends rather in depth than in area, the limpet-shell shape is produced. In the flatter variety the resemblance to an oyster-shell is increased by the surface having a more or less flaky appearance, and in the more prominent kind the limpet-shell appearance is often enhanced by the crust being studded with a number of small obtuse nodules. Sometimes the thickest of these crusts are somewhat constricted at the base; this may be noticed occasionally when they affect the neighbourhood of the face. The ulcers are large (attaining sometimes the size of a crown-piece), are irregularly rounded, and have perpendicular edges; their floor is formed either of flabby granulations or of a tenacious slough-like layer, and they discharge a fetid, sanious, plastic pus: they are deeper under the thicker crusts. The number of the rupial patches varies greatly; thus even in a long-standing case there may have been but two or three, while in other instances their number is very considerable.

In some cases the disease assumes a serpiginous character, spreading centrifugally over a large extent of surface. Its mode of progression and the general appearance which it presents under this condition will be found described under the head of "ulcerating tubercular Syphilides," with this difference only, that in this, the rupial affection, the outer ring is formed by purulent blebs instead of tubercles. The cicatrices left by the ulcers present the various appearances described in the account of the ulcerating tubercular Syphilides.

The rupial Syphilide may occupy any region, and is sometimes spread over the greater part of the surface, but it affects preferably the lower limbs or the head (scalp or face). It is met with usually in debilitated or cachectic subjects, especially in those in whom the general condition known as the syphilitic cachexia has become confirmed.

It rarely appears earlier than a year or two after primary Syphilis, and may not be developed till several years have intervened. It is usually of long duration, and if not cut short by suitable treatment may even last many years.

Prognosis.—This will be unfavourable either as regards immunity from permanent local effects or the prospects of speedy recovery. The eruption invariably leaves behind it conspicuous scars, which will be disfiguring in proportion to the depth or the area of the ulcers th succeed, and the period of recovery will be remote according to t degree in which the general health is affected.

Diagnosis.—Syphilitic Rupia, or as it should more properly termed, rupial Syphilide, is so peculiar in its appearance as to scarcely liable to be mistaken for any other cutaneous disease. T only disease that at all resembles it is Lupus, but the pale granul tions that form the floor of the ulcers of Lupus and the oth diagnostic characters mentioned elsewhere as distinguishing Lup from the tubercular Syphilides, are sufficient to prevent an error this kind.

Tubercular Syphilide, or "syphilitic Lupus," as it is sometim termed, is a not uncommon form of syphilitic cutaneous disease. is one of the later phenomena of constitutional Syphilis, and does n usually appear until after the occurrence of some of the earlier man festations of the syphilitic diathesis. It may present itself for th first time many years after contagion, but at any period is always very persistent affection. It is one of the "tertiary" or more i veterate forms of cutaneous Syphilis.

Tubercular Syphilide is of two kinds, the one, usually of earli development than the other, very rarely leads to ulceration, while the later and graver kind ulceration is invariably produced.

The former kind comprises two varieties, *the clustered* and *t scattered.*

The latter kind is divided, according to the character of the ulce induced by it, into *the perforating* and *the serpiginous* varieties.

The clustered non-ulcerating variety is the commonest form tubercular Syphilide. It appears in the form of small, rounded, pr minent, reddened tumours. These gradually acquire a dusky copper red colour, and their surface becomes tense and shining; their size vari from that of a pea to that of a nut. They are generally disposed small circular or elliptical rings, which, though sometimes complet are more frequently interrupted in two or three places, or even eac of the component tubercles may be perfectly distinct from the res The area enclosed by the circle, as well as the intervals where th continuity of the latter is broken, is sometimes occupied by a supe ficial, tawny-brown, slightly desquamating cicatrix. In other instanc the tubercles are disposed in small irregular groups; when th arranged they are smaller, harder, and more prominent than whe in the annular form. They terminate ordinarily, although not i variably, in resolution, their surface losing its polished appearan and becoming slightly scaly, their substance getting soft and flabb and their prominence gradually diminishing till at last a depress indelible cicatrix is left, which retains for some time a tawny-brow colour. This cicatrization is the result of the interstitial absorption the disorganised tissue, and takes place without breach of surfac Sometimes, however, the tubercles ulcerate and become covered wi dark-green crusts, under which are formed cicatrices of the kind ju described. This variety of tubercular Syphilide appears most commonly on the face (forehead, nose, mouth, or chin); on the forehead it has received the name *corona veneris.* It runs a chronic course, lasting usually for several months.

The scattered non-ulcerating variety occurs in the form of small, rounded, or oval tumours, varying in size from that of a pea to that of a hazel-nut. They are sprinkled thickly or thinly, but in either case pretty equably, over the region occupied by them. Their colour, at first a deep red, soon changes to a tawny coppery-red. They are of a pretty firm consistence. Their surface is shining, and they have a peculiar infiltrated semi-transparent appearance. After a time their somewhat flattened summit becomes covered with a thin, whitish, adherent scale, which gradually peels off at its circumference, and at length becomes completely detached. After this the tubercle begins to get flabby and to diminish in size, and finally disappears leaving a depressed, brownish, indelible scar, the colour of which slowly fades away. In some instances the cicatrix altogether becomes after a time so faintly marked that it may be a matter of some nicety to establish its existence. This variety is met with principally on the face, but it occurs on the trunk and arms. Its duration extends usually over several months.

The perforating ulcerating variety appears in various situations, but usually on some part of the face, in the form of two or three clustered tubercles. These are of comparatively large area, varying in size when fully developed from that of a threepenny-piece to that of a shilling, or even of a half-crown. Their elevation above the level of the skin is but slight in proportion to their size. They are however deeply rooted. They are rather subcutaneous swellings than actual tubercles. At first they are hard, firm, and elastic, and the skin covering them is reddened, the redness gradually acquiring a coppery character. After a time the tumour softens, the coppery-red skin becomes livid and thinned, and at length ulcerates. A black, dry, thick, rugged crust soon forms over the sore. On detaching the scab a deep ulcer having perpendicular edges (as if it had been cut out with a punch) and a grey slough-like floor is laid bare; from this a fetid, sanious, plastic discharge exudes, which, concreting speedily, reproduces the crust. Under this the ulcer, without extending notably in area, gradually increases in depth, eating its way slowly through whatever tissues may lie next beneath it, even cartilage or bone. The margin of the ulcer is surrounded by a coppery-red areola. The duration of the affection is always long. Its progress may become arrested either spontaneously or under the influence of treatment; the ulcer then becomes cleaner, florid granulations take the place of the grey slough-like substance that formed its floor, it becomes shallow, and at length cicatrizes. The scars left by this variety of Syphilide are usually somewhat depressed below the level of the skin; they may either be polished and even, or puckered and intersected by raised tendinous bands, which meeting one another give rise to hard tendinous knots.

When recent the scars are of a livid coppery colour, this changes slowly to a dull-brown; which becoming gradually fainter and fainter at length disappears, and a dull-white indelible scar is left.

The serpiginous ulcerating variety begins by red, hard, shining tubercles, which gradually assume a coppery tint; after a time they soften, their summits ulcerate and become covered with hard, dark-coloured, uneven crusts, and on detaching one of the crusts a shallow but abrupt ulcer is disclosed, having a grey pultaceous floor and exuding a sanious pus, which soon reproduces the crust: under this by degrees the ulcer granulates up and gets glazed over with a livid-brown, tolerably smooth cicatrix, which in process of time becomes blanched.

While the tubercles that first appeared are undergoing these changes, fresh ones are developed close around them, and while they in turn are following a similar course a third crop is appearing immediately beyond. In this way the disease extends centrifugally, its margin being formed by tubercles with, immediately behind them, a broken row of crusts, while the cicatrix in the interior is gradually being increased in area by successive additions to its circumference. Usually, however, the widening circle of active disease becomes broken and incomplete, owing to portions of it healing, and the rest of it continuing to spread, the patch of altered skin, instead of remaining discoid, acquires an irregular shape. The broad cicatrices left by this affection are in some cases smooth and even, though usually somewhat pitted; in others, however, they are puckered, and resemble the scars of severe burns. This variety of tubercular Syphilide occurs on the back, the chest, the face, and on the limbs in the neighbourhood of the joints. There are often several patches of it at a time.

Prognosis.—The different varieties of tubercular Syphilide are not all of them equally chronic, nor does each affect the general health in the same degree, nor are they similar in the matter of leaving behind them permanent scars.

In either of these respects the prognosis of the ulcerating will be less favourable than that of the non-ulcerating varieties.

As regards chronicity, the duration of either of the non-ulcerating varieties extends usually over a range of several months, while the ulcerating kind may be prolonged over several years.

The scattered non-ulcerating variety produces the least permanent defacement, while the perforating ulcerating variety is the worst in this respect; indeed the large destructions of tissue produced by the latter, and its preference for the face, cause sometimes the most hideous disfigurement: thus one of the alæ, or even the greater part of the nose, may be destroyed, the mouth may be greatly deformed, an eyelid may be wanting, the frontal bone may be perforated, or even, as I have had occasion to see, a large part of the calf of the leg may get eaten away. The same variety too is the most chronic of the tubercular Syphilides, and is the least amenable to treatment. It is moreover attended with a more marked deterioration of the general health than any of the others.

The serpiginous is neither so persistent, so obstinate, or so grave an affection as the perforating variety; still it leaves most extensive (although superficial) permanent scars, and when copiously developed it is apt to impair considerably the general health.

Diagnosis.—The *scattered non-ulcerating* variety may possibly be confounded with Acne indurata, but in the latter there is usually an oily condition of the neighbouring skin. The syphilitic tubercles are more superficial than those of Acne, which are more or less deeply embedded in the substance of the skin; the latter commonly suppurate, and on puncture may always be ascertained to enclose a small collection of sebaceous matter in their centre. The scars left by them are elevated and linear, those left by the syphilitic tubercles are on the contrary rounded and depressed. The extent of surface occupied by the eruption of Acne is usually more limited, and the nodules are of a somewhat purplish, or at the least of a crimson red, whereas the area of the syphilitic eruption is comparatively extensive, and its nodules soon acquire a coppery hue.

The *clustered non-ulcerating* variety, to which the name of "syphilitic Lupus" more especially belongs, is very apt to be mistaken for (scrofulous) Lupus properly so-called, and this more particularly when it affects the nose or the cheek, which are the common situations of Lupus. But the tubercles of Lupus are commonly of a purplish hue; they are more flabby and have a duller surface—that is to say, they want the tense shining appearance of the syphilitic tubercles; they are often accompanied by a puffy state of the neighbouring subcutaneous cellular tissue. The ulcers produced by the scrofulous disease either have undermined edges or are ill defined at their margin, and their base is composed of pale flabby granulations, whereas those of the syphilitic eruption have clean-cut abrupt margins and a slough-like floor.

The scars of Lupus are irregular, contracted, and often ridgy, while the syphilitic scars are circular, even, and depressed.

Lupus appears generally during the juvenile age, the tubercular Syphilide during adult age, and although the latter may fairly be considered a chronic eruption, its chronicity is as nothing in comparison with that of Lupus, which progresses by almost imperceptible gradations, and endures for many years.

When arranged in rings the clustered variety may be possibly confounded with Psoriasis circinata, but in the latter the reddened ring is more continuous, its surface is rougher and duller, and it is covered with scales, which exhibit a nacreous sheen.

The *perforating variety* of the tubercular Syphilide can scarcely be mistaken for any other cutaneous disease.

The *serpiginous kind* is liable to be mistaken for a phagedænic chancre, but with the latter no symptoms of *constitutional* Syphilis are present, the ulceration extends more rapidly, and spreads from the ordinary situation of the chancre (the neighbourhood of the genitals); moreover, the sore is inoculable.

Treatment of the Syphilides.—The remedy *par excellence* for constitutional Syphilis is mercury; attempts have been made from time to time to discover some better remedy, and now and then it has been the fashion to enlarge on the dangers and disadvantages of employing this drug in the treatment of Syphilis. It has been said that the worst results of so-called tertiary Syphilis were due rather to the remedy than to the disease, and it has even been proposed that Syphilis should be left altogether alone as being a very innocent affection in itself, provided it were not meddled with. Nay, some have gone further even than this, and have steadfastly advocated that the syphilitic patient should be, if possible, still further infected by the repeated inoculation of chancre-pus at various parts of his body, and that this process should be persevered with until he became so thoroughly syphilitic as to be quite well! This doctrine (of "syphilization") has had some illustrious advocates. But notwithstanding all that has been urged against the use of mercury in Syphilis, it still holds its ground as the most valuable of anti-syphilitics. It may be confidently stated that no means of treating Syphilis that has yet been proposed can compare with mercury in efficacy and in rapidity of action, and that no remedy is attended with less disadvantages provided it be used with discretion. At the same time it must be admitted that, although the best, it is by no means a perfect remedy for Syphilis.

It is useless prescribing a course of mercury for a person affected with an indurated chancre in the expectation of securing him by such means immunity from all furthur manifestations of constitutional Syphilis, and it is equally futile to persist in administering small doses of the drug after the total disappearance of all perceptible symptoms of constitutional Syphilis in hope of "thoroughly eradicating the syphilitic virus" by this process. Either of these precautions is about as idle as the no less popular plan of continuing to administer arsenic in cases of (simple) Psoriasis long after the eruption has disappeared with the view of preventing any future attack by removing in this way any "lurking traces" of the disease that may remain in the system. For clinical experience shows very clearly, both in the case of the acquired (syphilitic) diathesis as well as in that of the congenital diathesis on which Psoriasis may be assumed to depend, that this anticipatory medication is valueless as a means of warding off future manifestations of the constitutional taint.

In the administration of mercury as an anti-syphilitic it is important to bear in mind that it is far better to give too little than too much.

In the earlier and more tractable manifestations of constitutional Syphilis, such as the papular and squamous Syphilides, it will usually be found, if the drug be administered in moderate doses, that the eruption will begin to fade very perceptibly before the gums begin to be affected, and in such cases it will rarely be necessary to push the remedy to the extent of affecting the gums at all. So long as the eruption continues to fade the drug should be withheld until the stationary condition of the eruption again demands its use. Indeed, in all cases it is preferable to fall back on the eruption as our guide in regulating the use of mercury, rather than to push the use of the remedy to the unnecessary extent of affecting the gums, in order that they may serve as an indication of its effect.

Accordingly, in the tertiary manifestations of Syphilis, which under any circumstances cannot be made to disappear so speedily as its secondary and more tractable symptoms, mercury should be given in still smaller doses, and greater patience should be exercised in waiting for the first appearance of a change for the better in the character of the eruption. When improvement, however, has once commenced, the condition of the eruption is the indication to be obeyed, provided that it be kept in mind that a far more gradual progress than in the case of the earlier Syphilides is the best result that can be obtained.

As to the form in which mercury should be given—that is to say, what compound of it should be employed, and as to the way in which it should be introduced into the system, whether by the skin (inunction, fumigation), by the nostrils (insufflation), or by the mouth (inhalation, pills, mixtures)—opinion is somewhat divided; but there appears to be no sufficient reason why the usual and most convenient way of taking remedies, viz., by swallowing them, should be deviated from when mercury is employed as a remedy for constitutional Syphilis, and the balance of opinion is in favour of this method of introducing the drug.

It is equally in favour of choosing the green iodide as on the whole the most suitable compound of mercury for use in this way. The dose of the green iodide, in the case of the earlier Syphilides, should be from a quarter of a grain to half a grain given thrice or twice a day in the form of a pill. In the later Syphilides half this dose is sufficient. Should any irritation of the stomach or bowels be occasioned by the pills, the addition of a small quantity of opium (a quarter of a grain to each pill) should be made.

From the foregoing it will be evident that the author considers mercury to be a remedy of great value in the treatment of tertiary as well as of secondary Syphilis. Many, however, while fully admitting the value of mercury in secondary Syphilis, consider that the remedy *par excellence* for the tertiary or later manifestations of the disease is the iodide of potassium, and that mercury in such cases is always useless and sometimes harmful. The iodide of potassium is doubtless a remedy of great value in tertiary Syphilis, but it is as an adjunct to and not as a substitute for mercury that it should be used. It is especially of value in relieving the *pain* of constitutional Syphilis, secondary as well as tertiary. It allays the wandering rheumatic pains that often accompany the earlier Syphilides, and is of equal efficacy in the fixed aching (osteocopic) pains in the bones, and in the darting (neuralgic) pains in the temples, that accompany the later phenomena of Syphilis.

It is also of value in promoting the resolution of (secondary) syphilitic nodes, as well as in favouring the absorption of the plastic deposits (cutaneous and other) that are so frequently met with as tertiary symptoms. It may be given in doses of three to ten grains thrice daily. The iodide of iron is with many a favourite remedy in tertiary Syphilis, and it is a valuable adjuvant to mercurial treatment in cases where there is much pallor and well-marked cachexia.

Cod-liver oil is useful in similar cases.

Sudorifics are believed by many to assist the action of mercury in Syphilis; accordingly the compound decoction of sarsaparilla taken warm is a favourite addition to the usual treatment. The same idea is embodied in the plan of treating Syphilis by mercurial fumigations, or by warm-baths containing a small quantity of corrosive sublimate in solution. The administration of simple vapour-baths is by some preferred to the internal administration of sudorifics.

Whatever may be the theory on which sudorifics are adopted (whether it be that by such means the "syphilitic virus" is sweated out of the system, or that by repeatedly determining a flow of blood to the skin the action of the mercury is specially directed to the situation of the eruption), the use of them does not appear either to hasten the disappearance of the eruption or to insure the patient against a relapse of it.

It is almost unnecessary to insist on the advisability of the patient's taking care of himself while he is under a course of mercury, or to say that he should avoid strong exercise, late hours, and exposure to wet and cold, and should adopt a moderate and unstimulating diet.

When the health has been much shattered, a short preparatory course of treatment may be advisable before specific treatment is entered on, such as the repeated administration of mild aperients, a nourishing and stimulating diet, a course of tepid salt baths, quinine, &c.

In cases where mercury has been taken on several previous occasions benefit may often be obtained by the iodide of potassium given alone. The reason of this is that after repeated courses of mercury a certain portion of the drug often gets deposited in the metallic state in some of the tissues, notably in the liver, spleen, skin, and bones, where it may remain inert for a considerable time. The solvent action of the iodide of potassium again introduces it into the circulation, and its specific effects again become manifested.

Regarding local treatment, many authorities hold that it is useless in the management of the Syphilides, and some advocate that even the deep ulcers of tertiary Syphilis should be allowed to heal by scabbing under the influence of constitutional treatment without means being employed for healing the sores by the direct application of remedies to them. These views, however, are not supported by the facts that have come under the author's observation; he has found that it is possible to heal even extensive eruptions of tertiary Syphilis by the use of local remedies only, and that too without any evidence presenting itself, either during or after treatment, of the system generally being put under the influence of the remedies applied. He is therefore justified in holding that local remedies are a considerable aid to constitutional treatment.

In the earlier Syphilides it will be sufficient to wash the skin well with soap and warm water daily, in order to remove the exudations that may interfere with the direct contact of the remedy with the diseased skin; but in the later Syphilides the incrustations should be softened by the application of poultices, and the ulcer cleansed by washing it with thin gruel and afterwards dried with a piece of soft lint before applying the remedy.

In the earlier Syphilides one of the best applications is an ointment of the nitric oxide of mercury, the oxide being prepared in the wet way, and used in the proportion of gr. xv., or ℈j or even ʒss to the ounce of simple cerate. The later Syphilides require a more stimulating application, and the addition of a small quantity of the red iodide of mercury (gr. j.—v. to the ounce) should be made to the ointment just described. Iodoform employed as ointment of various strengths, or as powder diluted or not with varying proportions of starch-powder, is a valuable local application in the later Syphilides.

In infantile syphilis the best general treatment is the administration of a grain of grey powder every other day. If there be much emaciation cod-liver oil should be given also.

The local treatment of infantile Syphilis should consist in the application of an ointment of oxide of mercury (the oxide made in the wet way) containing five grains of the oxide to an ounce of simple ointment.

The same ointment should be applied to mucous tubercles, and they should be painted over every second or third day with tincture of iodine, the ointment being of course previously washed off with soap and water.

Cauliflower vegetations are best healed with escharotics in the first place, and the application of oxide of mercury ointment afterwards.

Nitric-acid, taken internally, has been praised as an agent in the treatment of the Syphilides, but its advocates consider it useful only in the case of the earlier Syphilides; its fame is due probably to the fact that the earlier Syphilides have a comparatively short duration, and disappear spontaneously after a certain time; at all events this may be said, that its effect, if it have any, is far more gradually produced than that of mercury.

Sarsaparilla has been vaunted as a remedy for constitutional Syphilis, and enjoys a high popular repute in this capacity; but it is quite inert.

SECTION X.

The animal-parasite diseases of the skin are Scabies and Phthiriasis.

SCABIES.

Synonyms.—The itch; the Scotch fiddle;[1] The yuck;[2] La gale; La gratelle; Die krätze; Scabrities; Psora.

Definition.—Scabies may be defined as that morbid condition of the skin that results from the presence of the *Acarus scabiei* in the substance of the epidermis.

Causes.—Scabies is most frequently met with amongst the poor, but the middle and upper classes are not altogether exempt from it. It is commoner with children than adults, and with men than women. Its only exciting cause is the presence of the *Acarus scabiei* in the substance of the epidermis.

The mode by which it is communicated is by the transference of the ova of the acarus from one person to another.

Description.—The disease usually makes its appearance in from ten days to a fortnight after it has been communicated. It commences with itching. This is at first limited to the hands and forearms and the lower part of the belly and upper part of the thighs, but it soon becomes general. The itching is at first felt only towards the evening. The eruption of Scabies, which varies in character, will be best described as different diseases of the skin which may be caused by the *acarus*. These are as follows:—

A Pruriginous Eruption.—This is the most frequent of all. It occurs in the form of minute papules, sprinkled loosely over the surface of the skin (not clustered together as in Lichen), exuding from their tops minute drops of serous blood, which concrete into small reddish crusts. The exudation is due, as in Lichen, to the excoriation of the tops of the papules by the scratching of the patient. This condition of the skin is to be looked for chiefly on the palmar aspect of the forearms, on the abdomen and on the anterior and inner surfaces of the thighs.

A Vesicular Eruption.—This appears in the form of small rounded elevations of the cuticle, containing clear lymph, scattered loosely over the surface of the skin, varying in size from that of a pin's-head to that of a split-pea, or even larger, and surrounded often with a rosy-red areola of inflamed skin. These vesicles occur on the hands and feet, but more especially on the hands, where they are to be met with chiefly on the backs of the webs of the fingers, on the lateral surfaces of the first phalanges of the fingers, on the back of the thumb and of the web of the thumb and on the front of the wrist. The vesicles are not so constant a symptom of the presence of the itch-insect as the papules. They are absent in about twelve per cent. of the cases of scabies.

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[1] This time-honoured joke against the inhabitants of North Britain is thus alluded to by Coleridge in his celebrated poem "The Devil's Walk":—

"He took from the poor,
And he gave to the rich,
And he shook hands with a Scotchman,
For he was not afraid of the ——"

[2] "The yuck" is a Scotch synonym for Scabies.

A Pustular eruption.—The description that has been given of the vesicles will apply equally to the pustules as regards their shape, their size, their arrangement, and their situation; but besides occupying the situation common to themselves and to the vesicles, the pustules are to be found also on the nates. They differ from the vesicles only in being opaque and yellowish instead of exhibiting a pearly transparency, and in being surrounded with a more decided inflammatory areola.

In addition to the above, eruptions having all the characters of *Eczema* or of *Impetigo* often form a part of the disease. These are to be observed usually about the wrists and ankles, in the flexures of the elbows and knees, and in the female on the breasts. *Furunculi* sometimes occur on the nates, and the author has occasionally met with cases of Scabies where an extensive *urticarious eruption* (although the patients had never experienced an attack of Urticaria before) formed the most prominent feature of the disease.

Besides the above-detailed eruptions, there is another symptom of Scabies which, though less obvious than any of the preceding, is of infinitely greater importance, since it is not only most constantly present, but, once recognised, is pathognomonic of the disease. The symptom referred to is the track left by the female acarus in its passage through the substance of the epidermis—the *acarian furrow* as it has been termed. This presents the appearance of a curved, dotted line, under the surface of the epidermis, varying in length from the thirtieth to the third of an inch, and assuming the form of a comma, of a horseshoe, or of the letter S. It may be either white or of a greyish colour. At one extremity of the furrow is a minute, rounded, opaque, white elevation, the "*acarian eminence;*" from this, with a little address, the acarus itself may be extracted on the point of a pin.

Scabies rarely attacks the face or scalp.

Prognosis.—Scabies, if not submitted to suitable treatment, may continue indefinitely, but under the influence of appropriate remedies speedily disappears.

Diagnosis.—This is of unusual importance in this disease on account of its extremely contagious character.

In no other eruption is any mark on the skin produced that at all resembles the acarian furrow. To the practised eye the discovery of an acarian furrow is as conclusive a proof of Scabies as even the extraction of an acarus from the end of the furrow and an inspection of the insect through the microscope could be. In the adult the acarian furrows are to be sought for in the epidermis of the hands; but in the infant they are as visible on the feet as on the hands.

But, independently of the acarian furrow, there are certain peculiarities of Scabies which enable it to be recognised without any difficulty, in at all events the majority of cases.

The existence of a pruriginous eruption on the inner surfaces of the forearms, on the abdomen, and on the anterior and inner surfaces of the thighs, and a discrete vesicular eruption of the hands and feet, or a discrete pustular eruption of the hands, feet, and nates. A confluent vesicular or pustular eruption of the flexures of the knees and elbows, of the front of the wrists, of the folds of the groins, and of the hollows of the axillæ.

An assemblage of such symptoms, or even of the first two of them, is usually enough to go upon, and a union of the first two occurs in most cases of Scabies.

Still it will sometimes happen that the acarian furrow is not to be met with; when this is the case it will generally be found (in cases which turn out to be nevertheless Scabies), that the patient has had previous imperfect sulphur-treatment. Whenever a case presents itself that has not had previous specific (sulphur-ointment) treatment, and yet presents no appearance of an acarian furrow under the scrutiny of a practised eye, presumption is strongly in favour of the case not being one of Scabies.

The pruriginous eruption may sometimes resemble prurigo senilis. The vesicular eruption may appear like eczema of the hands. The pustular eruption may resemble impetigo of the hands, or ecthyma of the nates, and the patches of eruption in the flexures of the joints, may resemble either eczema or impetigo.

Prurigo senilis, however, occupies chiefly the back of the shoulders, the outer surfaces of the upper arms, the loins, and the *outer* surfaces of the thighs. It *never* affects the fingers. The itching occasioned by it is far more severe than that of Scabies. Its minute scabs are larger and of a darker colour than those which tip the papules of Scabies. The *pediculus corporis* is to be found on the under-clothing.

In *eczema* of the hands the *dorsal* surfaces of the fingers are more affected by the eruption than their lateral surfaces.

Impetigo of the hands usually occurs in circumscribed patches.

It may be stated generally, that if an eruption has lasted two or three weeks and still remains confined to one region, it is not likely to be Scabies, which is an eruption that soon becomes general.

Scabies generally commences on the hands or forearms; if the patient relate that his eruption began elsewhere, the probability is against its being Scabies.

Treatment.—Various remedies have been recommended for the treatment of Scabies. Stavesacre, iodide of sulphur, essential oils, mercury, iodide of potassium, benzol, and a host of others; but the remedy that is generally employed is sulphur, and this substance has long been considered as the most efficacious agent that can be employed in the treatment of Scabies. It enjoyed this repute long before it was known that the efficacy of sulphur as a cure for the itch, depended on its poisonous effect on the acarus, and its reputation as an "itch-specific" is as great in the present day as it ever has been. Several ways of administering it have been proposed; some give it internally, some internally and externally, but most are agreed that it is necessary only to administer it externally. As to the extent of surface over which it should be applied, differences have arisen; some authorities consider that its application should be limited to the parts affected with eruption, others think it should only be applied to the regions where the acarus is visible, viz., the feet and hands. But the balance of opinion is justly in favour of applying the sulphur to every part of the skin that is liable to be affected by the eruption of Scabies. The mode of applying the sulphur has been a matter for discussion; some prefer it as a fumigation and some as a lotion, but most apply it in the shape of ointment. The composition of the ointment has been a subject of debate. The Unguentum Sulphuris of the late London Pharmacopœia was composed of one part of sublimed sulphur and two of lard; this was an unnecessarily strong compound. The Unguentum Sulphuris compositum, of the same Pharmacopœia, contained a fifth part of sublimed sulphur, a fifth part of soft soap, a sixteenth part of white hellebore, and a two hundred and fortieth part of nitrate of potash; this was too irritating a compound. The Unguentum Sulphuris of the British Pharmacopœia consists of one part of sublimed sulphur, and four parts of benzoated lard. These are suitable proportions, but the ointment is more efficacious if precipitated be substituted for sublimed sulphur. The former is in a much more finely divided state than the latter, and the old objection to the employment of precipitated sulphur, viz., the very extensive adulteration of it with sulphate of lime (to the extent even of 50 or 60 per cent.), is no longer valid since the precipitated sulphur is now sold in a state of almost perfect purity. The length of time required for treatment by ointment made with the sublimed sulphur, and the relapses that occasionally take place even after a prolonged use of it, have led some observers to seek after means of rendering it more speedy and certain in its action. The plan which has met with most favour, is to mix with the ointment a small quantity of the subcarbonate of potassa; about a drachm to the ounce of the unguent. sulph. P.B. is the proper proportion. The potash, by softening the cuticle, promotes the action of the ointment. A preferable plan, however, in the estimation of the author, is to use a small quantity of the hepar sulphuris in place of the subcarbonate of potash. A very efficacious remedy is the "Balsam of sulphur," a solution of sulphur in warm olive oil.

Glycerine is used in the place of lard as a vehicle to mix the sublimed sulphur with, but lard is for many reasons a far better substance for the purpose than the at present somewhat overrated glycerine. Whatever form of sulphur ointment be used, the following rules should be observed in the treatment of Scabies.

Before applying the ointment, every part of the body excepting the head and face should be thoroughly washed with soap and water. For infants and young children, mild (neutral) soap should be used, but for adults, strong (alkaline) soap is necessary, the "*best*" "soft soap" answering the purpose very well.

After a thorough soaping of his skin, the patient should take a warm bath, remaining in it for about half-an-hour.

On emerging from his bath he should be thoroughly dried with warm towels, and then the ointment should be rubbed well in over every part of him, excepting only his head and face.

After he has been thus thoroughly anointed, he should put on linen or cotton, not flannel, underclothing next his skin, and keep the same underclothing on for two days and two nights without change. At the end of this time the ointment should be reapplied in the same manner, and with the same preliminaries as before.

If the ointment used be an efficacious one, and the treatment be energetically pursued, two or three applications will suffice to eradicate thoroughly the disease.

When Itch has broken out in a family it is important that all the members of the family who have contracted it should be treated simultaneously, and that those who have apparently escaped contagion should be watched for at least a fortnight after treatment has been commenced with the others. For it should be remembered that the disease has its period of incubation; and that the freedom from eruption of a person who has been very recently exposed to the contagion of Scabies, is no proof that he has not contracted the disease. It will be obvious that the popular plan of submitting to treatment only such children of a family as are suffering from the complaint, and then after they are well, of finding out that the disease has become sufficiently developed in another child to demand interference, is merely to perpetuate the disease indefinitely in such a family.

PHTHIRIASIS.

Synonym.—Louse-disease; Maladie pédiculaire; Die Läusesucht; Morbus pedicularis.

The eruption produced by this parasite has already been described (under the heading of Prurigo) by the title of *Prurigo senilis.* It remains to be stated here that the pediculus corporis is not always easy to be found, and this is probably the reason why this parasite is not generally supposed to play the important part in the causation of cutaneous disease that the author's researches have led him to credit it with. On stripping a person affected with Prurigo senilis, it would not occur to any one who was unaware of the fact that the disease was produced by a pediculus. The pediculi are rarely numerous enough to arrest the eye, and more than this, even a careful scrutiny of the skin, including in the survey the parts of it that are most affected with eruption, will, in the majority of cases, fail to detect the presence of the parasite; not even a *nit* is to be seen on any part of the skin or on any of the hairs growing from it. Now the pediculus capitis and the pediculus pubis—in cases when the insect itself is not easily to be found—may always be readily detected by means of the nits attached to the hair of the part they inhabit. It is not to be wondered at, then, that the part played by the pediculus corporis should have been so often overlooked. The parasite lives, not on the skin, but on the underclothing,[1] and it deposits its eggs neither on the skin nor on the hair, but on the underclothing. It is on the inner surface of the undermost article of clothing, whatever it may be, that the parasite is to be sought for, and here it is not always very easy of discovery. As already stated, the pediculi are rarely to be found in numbers; a very few of them are capable of causing very severe irritation. A careful investigation of the patient's shirt may lead to no result, and yet his disease may be due solely to the pediculus. The parasite nestles in the "gathers" or folds of the shirt; these are most numerous in a man's shirt at below the back of the collar of the shirt, and hence it is that in men the disease is usually most severe on the back of the shoulders, and always more severe on the shoulders than on the chest. In women (whose under-garments are arranged differently) the eruption is commonly as severe on the breast as it is on the back of the shoulders. At and below the waist, where the shirt is again thrown into folds, the pediculus again establishes himself, and accordingly the loins and upper part of the thighs are also common situations of the disease. The nits of the pediculus are also to be found in the same situations, but occasionally a larger cluster of them is to be found at some other part of the shirt.

The pediculus corporis affects men and women indifferently, and is to be met with in persons of all ages, but it more commonly infests adults than children, and is commoner with aged persons than with adults.

PEDICULUS CAPITIS.

Synonyms.—Head-louse; Pou de tête; Kopflaus.

This parasite occupies only the hairy scalp, and gives rise to eruptions of the scalp and of the neighbouring skin. It lives in the hair. The pediculus corporis, as we have just seen, lives in the under-clothing. But although it inhabits the hair of the head, it never invades the hair of the whiskers, beard, moustache, eyebrows, axillæ, or pubes, all of which situations, however, are liable to become infested by another kind of pediculus. The favourite habitat of the Pediculis capitis is the *occipital* part of the scalp; here it is always to be found in greater numbers than at any other part of the scalp, and here it is (in cases where the nature of a scalp-eruption may become a question) that evidence of the presence of the pediculus should be sought for. In cases where the hair is thick and the pediculi being few are therefore not immediately visible, their presence may at once be discovered by the existence of *nits* (the ova), sticking in numbers on to the hairs. The nits are readily to be distinguished from scurf by their oval outline, and their firm attachment to the shaft of the hair. They are to be found deposited in rows on the hair, and are sometimes arranged so closely as to give the hair a moniliform appearance. In children the Pediculus capitis is of far more common occurrence than it is in grown-up persons. In the child the eruption produced is generally *impetigo* of the scalp. Some writers who admit that the Pediculus capitis may be concerned in the production of impetigo of the scalp offer no suggestions for distinguishing between the impetigo arising from this cause, and constitutional impetigo, and are evidently unaware of any difference existing between the one and the other.

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[1] It is, of course, not to be understood from this that it does not *feed* on the skin. It lives upon blood just as the flea does.

Some appear to think that impetigo granulata is the particular eruption that is caused by the head-louse. The author's researches have, however, shown him that the essential difference between the constitutional and the parasitic eruption is, that while the former almost invariably affects the anterior half of the scalp more severely than the posterior half, the parasitic eruption is always more developed over the occiput than it is at any other part of the scalp.

It must be remembered, however, that the presence of pediculi on the head of a child by no means invariably produces an eruption of impetigo. It is not a rare thing in public practice to meet with children whose heads are so thoroughly infested with pediculi as to lead to the inference that the latter are inhabitants of old standing, and yet there is often no eruption of the scalp except a scanty pruriginous one; the patient states that he experiences scarcely any irritation, and his parents say that he scarcely ever scratches his head.

Pediculi are less commonly to be met with on the heads of grown-up persons than they are in children, and either in men or in women their presence less commonly provokes any eruption (further than a scanty pruriginous one). The author has observed that the eruption commonly produced in adults is *not* an eruption of the scalp. The portions of skin most affected are those that are covered by the depending hair of the scalp, and the eruption is abruptly limited above by the margin of the scalp, and below by the ends of the depending hair. The character of the eruption is generally purely lichenous (circumscribed lichen), but it sometimes, though very rarely, assumes the form of eczematous lichen.

The situations occupied by this lichenous eruption are the temples, the ears, and the upper part of the back of the neck.

PEDICULUS PUBIS.

Synonyms.—Crab-louse; Morpion; Filzlaus.

While the pediculus capitis may be considered as more especially the pediculus of children, and the pediculus corporis as the pediculus of old persons, the Pediculus pubis may be regarded as the pediculus of adolescents.

The pediculus corporis, as we have seen, attacks parts of the surface that are comparatively hairless, and appears to avoid those parts of the skin that are thickly covered with long hair.

The pediculus capitis and the Pediculus pubis, however, attack only such portions of the skin as are pretty thickly covered with hair. The pediculus capitis, as we have already learnt, limits itself to the hair of the scalp. Now all the rest of the longer hair of the body is the domain of the Pediculus pubis, viz, the pubic hair, the hair of the abdomen and chest, the hair of the axillæ, the beard, whiskers, moustache, eyebrows, and eyelashes. In the child the habitat of the crab-louse is limited to the eyebrows and eyelashes. A case of this kind lately came under the author's observation. The stronghold of the Pediculis pubis in the adult is the pubic hair; here it is always found in greater numbers than it is elsewhere, and here it is that it makes its first appearance.

The pediculus capitis, as we have seen, lives on the hairs, and deposits its eggs on the hairs; the pediculus corporis lives on the clothes, and deposits its eggs on the clothes. Now the Pediculus pubis lives on the skin and deposits its eggs on the hair. The flat crab-like insect lies closely attached to the skin, and if any attempts be made to detach him he immediately digs his claws into the epidermis, and anchors himself so firmly to the skin, that it requires the exercise of some force to pull him off. The pediculus capitis crawls about the hairs, nestling between them; accordingly we find its ova deposited as often near the free ends of the hairs as near their roots. But the ova of the Pediculus pubis, which keeps close to the skin, are always deposited on the hairs very near to their roots.

The eruption provoked by the Pediculus pubis is a pruriginous one. It is often so severe as to simulate prurigo senilis, but the situations occupied by the eruption, corresponding to the habitat of the Pediculus pubis, the discovery of that insect on the skin, and the presence of nits on the hair, will always suffice to avert any confusion of the one with the other.

Prognosis of Phthiriasis.—Louse-disease in either of its three forms may last indefinitely if unchecked by appropriate means, but if recognised and submitted to suitable treatment it may always be speedily remedied.

Treatment of Phthiriasis.—Various remedies are used, all having for their object the extermination of the pediculus. Amongst these may be mentioned the powder of staphisagria seeds, the powder of the flowers of the Pyrethrum album, or of the Pyrethrum roseum, olive or almond oil, sulphur in the shape of fumigation or ointment, mercury in aqueous solution, as a fumigation or in ointment, turpentine, the various essential oils, etc.

Of the "powder" of staphisagria seeds it may be said that it is at best but a clumsy contrivance. The seeds are incapable of being reduced to anything like powder. The most skilful drug-grin cannot produce with them any better result than a coarse meal, mu like linseed-meal, an utterly unmanageable application for the purpo unless, indeed, a poultice were made of it. Finding that the m contained a certain amount of oily matter, the author had the removed from a small quantity of the meal by percolation with eth and found that the meal was capable of being reduced into a fi powder. He employed this powder in several cases of phthirias and found it quite inert. On inquiring what proportion of oil had be extracted from the meal he found that it amounted to as much as on half (by weight) of the meal; on making trial of the oil suitably dilut with olive oil he found it as efficient as any remedy he has ever tri against phthiriasis. A cheap way of preparing the oil for applicati is to digest the seeds in melted lard and strain while hot. The filtra is an ointment of the seeds of stavesacre. Two drachms of the bruis seeds should be used to an ounce of lard.

The powder of the flowers of the Pyrethrum is in the autho experience a much less efficacious remedy than the stavesacre.

Olive or almond oil act only in a mechanical manner. By formi a film over the pediculus they occlude its spiracles, and so asphyxia it. Unless applied very abundantly they are but uncertain remedi Turpentine is objectionable on account of its strong odour and its ir tating effect on the skin.

The essential oils are of uncertain efficacy.

Sulphur is not nearly so efficient a remedy as mercury, which indeed on the whole a preferable remedy to any that have been me tioned. Those who are afraid to apply it over a large surface of sk for fear of producing its effects on the system can use the ointment the oil of stavesacre, which is scarcely if at all less efficacio but sometimes irritates the skin. But such fears are groundless; it not necessary to *rub in* the mercurial ointment; all that is needed that it should be lightly *smeared* over the affected regions, and there no occasion for using a strong ointment. The author, who has treat many hundred cases of phthiriasis by mercurial ointment has not in single instance occasioned any of the symptoms of mercurial absor tion. The preparations that he generally employs are the Unguentu Hydrargyri Mitius of the Dublin Pharmacopœia. An ointment co taining ten grains of the oxide of the mercury to the ounce, or citri ointment mixed with an equal quantity of lard.

SECTION XI.

The Vegetable-parasite Diseases of the Skin are—Tinea favosa, Tinea tonsurans, Tinea decalvans, Sycosis and Chloasma.

TINEA FAVOSA.

Synonyms.—The Honeycomb Ringworm; La Teigne (par excellence); Teigne lupineuse; Teigne jaune; Teigne à rayon de miel; Erbgrind; Honigwabengrind; Favus; Porrigo lupinosa.

Definition.—Tinea favosa, or (as it is more commonly called) Favus, may be defined as that disease of the skin which is occasioned by the presence of the vegetable parasite known as the *Achorion Schönleinii* in the substance of the epidermis and of the hair.

Causes.—(Predisposing.)—Childhood; poverty; the lymphatic temperament; the scrofulous diathesis; the masculine sex. (Exciting.)—The presence of the *Achorion Schönleinii.*

Description.—The disease begins with itching and redness of the affected skin and with a furfuraceous desquamation of the cuticle. At the same time the hair loses its polish and becomes more or less brittle.

Soon small, isolated, dry, yellow crusts, not larger than a pin's head, make their appearance. These, as they extend at their circumference and increase in thickness, become depressed at their centre; very often a hair passes through the middle of the depression. These sulphur-yellow "Favus-cups" are pathognomonic of the disease. They are generally pretty numerous and are commonly surrounded by an areola of inflamed skin. Their size does not usually exceed that of a split pea.

In this condition the disease has been called *Favus lupinosus* on account of the supposed resemblance of the small "cups" to the seeds of lupines. When sufficiently near to one another they may meet at their circumference, still retaining much of their original form. In such cases their rounded margin is pressed into a hexagonal shape, and the cup-like depression in the several crusts remaining, the surface bears some likeness to the cross section of a honeycomb. This appearance has given origin to the name of Favus.

After a certain time this honeycomb aspect is altered by portions of the cups getting detached, so that their depressions become obliterated. Their colour, however, remains unchanged. In this stage the incrustation forms large irregular yellow patches (*Favus scutulatus*).

Later still the crusts lose their yellowness and become white, friable, and uneven, so as to resemble very closely in colour and consistence, as well as in the conformation of their surface, the pieces of crumbling mortar that fall from old walls. In this state the disease is termed *Favus squarrosus.*

If in the early stage of the complaint one of the Favus-cups l carefully detached, there will be seen a smooth, shining, concave depression in the skin, somewhat red but without abrasion. At th same time it may be observed that the under surface of the cup convex, and is much smoother and of a deeper yellow colour than it free surface.

Coincidently with the development of the crust of Favus a furthe change beyond what has been already mentioned is undergone by th hair; the greater part of it falls off, and what remains loses colou and becomes short and woolly.

The head of a person affected with Favus exhales a peculiar fœti odour which has been variously likened to that of mice, of anima tissues undergoing maceration, or of the urine of cats.

Upon examination under the miscroscope the Favus-cups will b seen to be almost entirely made up of parasitic matter. This consist of (*a*) an amorphous homogeneous, finely granular *stroma*; (*b*) narro sinuous ramified tubules containing molecular granules (the *mycelium*) (*c*) broader (*sporoforous*) *tubules* containing minute rounded cells, o elongated cells placed end to end so as to give the tubules a jointe appearance; (*d*) *spores*, some free, others joined end to end like string of beads. The stroma and mycelium are found in greates abundance towards the lower (convex) surface of the Favus-cup, whil towards its concavity the cup consists chiefly of sporules. With th parasitic matter is a scanty admixture of epithelium cells.

If the diseased hairs be submitted to microscopical examinatio the filaments and spores of the *Achorion Schönleinii* will be foun also in the substance of the stem and of the knot of the hair as wel as in its root-sheath.

Besides the symptoms above described there are other phenomen which are to be regarded rather as accidental complications than a essential conditions of the disease. Thus not unfrequently the pustules of ecthyma or the crusts of impetigo are to be seen mingle with the Favus-cups; and in the majority of cases the head is at th same time found swarmed with lice.

Favus attacks ordinarily the hairy scalp, but it is to be found also o any part of the skin that is furnished with hairs, however rudimentar these may be. It occurs also on the nails. Wherever it present itself it exhibits the same characters.

Favus is in this country one of the rarest of the diseases of th skin. It is confined to no special period of life, but it appears mos commonly between the ages of six and ten. It affects especiall children of lymphatic temperament; the poor oftener than the rich Its only exciting cause is contagion.

Prognosis.—When taken early Favus is a curable disease, but afte it has existed for some time it occasions incurable baldness throug atrophy of the hair-follicles, and is apt to leave behind it extensiv cicatrices. When it has lasted for several years it produces genera pallor, emaciation, and debility.

Diagnosis.—In no other eruption are the characteristic spores and mycelium of the Achorion Schönleinii to be found in the substance of the hairs and of the epidermis. Favus may, however, be mistaken for Impetigo, Psoriasis, Tinea tonsurans, or Tinea decalvans.

However, in *Impetigo* of the scalp (the disease with which Favus is most likely to be confounded) the crusts are of a brownish rather than of a yellowish (as in Favus lupinosus) or a whitish colour (as in Favus squarrosus): they are not cup-shaped at the beginning, nor do they later assume a white powdery appearance. The odour exhaled by an impetiginous scalp, although often very offensive, has not that peculiar mousey quality that is characteristic of Favus. The hair is not so easily extracted. There are no bald spaces. The crusts are of a moister character. When there is (secondary) Impetigo complicating Favus, there is generally, at some portion or other of the scalp, an unmixed patch of eruption which exhibits, unmasked, the peculiar characters of Favus.

In *Psoriasis* of the scalp the incrustations are whitish but not powdery: they are stratified, and break up into scales, not into amorphous fragments. There are no cicatricial bald surfaces, the hair is not so easily extracted, and patches of psoriasis exhibiting their special nacreous sheen are to be found usually also over the knees and elbows.

In *Tinea tonsurans* there are no crusts and no absolutely bald patches. There are scurfy patches where the hair is broken off at the distance of about a line from the skin, but the scurf is neither yellow nor white but of a slate-grey colour.

In *Tinea decalvans* there are no crusts, no scurf even; there are simply smooth polished circumscribed bald patches from which a fine down may on close inspection be seen growing.

Treatment.—The local treatment of Favus should be commenced by cutting all the hair that remains on the scalp so short as to leave only half an inch of its growth. This is to be done with the double object of facilitating the removal of the crusts and of making the scalp ready for the process of epilation. The next step is the removal of the Favus crusts. This may be facilitated by a preliminary poulticing so as to thoroughly soften the crusts. But the application of lint soaked in dilute acetic acid, and covered over with oil-silk to retard the evaporation of the volatile acetic acid, answers the same purpose more readily. The acetic acid permeates the epidermic tissue in which the Favus matter is imbedded, and by softening and swelling up the epithelial cells, makes the whole mass soft and easy of removal. The aromatic smell of the acetic acid, by neutralizing the offensive smell of the Favus matter, makes its use preferable to the poulticing of the Favus crusts, which is practically a very disgusting process.

After as much of the softened crust as possible has been scraped off, the head should be well washed first with dilute acetic acid and afterwards with soap and water, and it is then ready for epilation. Epilation or pulling out the hairs from the scalp is a comparatively easy process in cases of Favus, since the hairs are much less firml implanted in the skin than they are in the healthy state, and the have not that extreme brittleness which renders epilation impracticable at first in the treatment of Tinea tonsurans.

The head of the patient may conveniently be rested in the lap o the epilator, or the patient may be seated while the epilator stand behind him. A common dressing forceps, furnished with a broad straight-edged bill, is the most convenient instrument to use. Th sensibility of the part about to be operated on should be diminishe by means of the ether-spray and epilation rapidly performed by seizin three or four hairs at a time with the forceps and pulling them ou in the direction of the slant of the hair at the part operated on Care should be taken *to pull* the hair *not to jerk* it out, since o account of its brittleness it is apt to break unless traction on it b gradually made. Several sittings will be required to effect the epilation of the whole of the scalp. After a portion of the scalp has bee epilated, it should be dealt with as directed in the description of the treatment, after epilation, of Tinea tonsurans. Sometimes a single thorough epilation, aided by assiduous application of the "parasiticide' remedies recommended in the treatment of Tinea tonsurans, is enough. But if, after the lapse of a few weeks the condition of the scalp appears to demand it, the epilation should be repeated.

The constitutional treatment of Favus consists merely in improvin the general health in any way that may seem necessary. As a genera rule cod-liver oil and steel are indicated.

TINEA TONSURANS.

Synonyms.—The Scurfy Ringworm; Boarding-school Ringworm Teigne tondante; Herpes tonsurans; Tinea capillorum; Porrig tonsurans; Herpes circinatus; Porrigo scutulata.

Definition.—Tinea tonsurans may be defined as that disease of th skin which is occasioned by the presence of the vegetable parasit known as the *Trichophyton tonsurans* in the substance of the epidermis and of the hair.

Causes.—(Predisposing.)—Poverty; childhood. (Exciting.)—Th contact of the spores of the Trichophyton tonsurans with the skin.

With regard to the causes above stated as predisposing to the diseas the first mentioned (poverty) is not more specially a favouring condition in this, than it is in other contagious diseases. The secon however (age) exercises a special and peculiar influence in th causation of Tinea tonsurans. Thus it is in children only that th disease is met with affecting the scalp, whereas both in children an adults it occurs on other parts of the surface; although in an situation it is commoner with children.

Description.—The disease presents a different aspect accordingly it affects the scalp or some other portion of the skin. When situate on the scalp it has been called distinctively Herpes tonsurans when occurring elsewhere it is generally distinguished as Herpes circinatus.

Herpes circinatus appears in the form of minute rounded, red patches. The rosy-red patches are slightly raised; their surface soon becomes rough and covered with a fine dry scurf; sometimes minute ephemeral vesicles appear on them. The patches rapidly increase in extent, retaining their original rounded outline; but as they spread they exhibit a disposition to heal at their centre, so that when they have existed for a few days they present the appearance of rounded patches of fading pityriasis bounded by a narrow lichenous ring. That is to say, their margin is decidedly raised, is of a rose-red colour, is covered with a tolerably abundant furfuraceous desquamation, and sometimes with ephemeral minute transparent vesicles, while the area inclosed by it is of a fainter (pink) colour, is scarcely, if at all, raised above the level of the healthy skin, is somewhat harsh, and is scantily covered by a very fine white scurf. At the centre of the patch the skin may approach still more nearly the healthy condition. There are usually several patches of variable size, some of which may attain a diameter of several inches. The patches are commoner on the exposed than on the covered portions of the body; thus in adults they generally occur on the backs of the hands, on the forearms, on the neck, or on the face; in women who are suckling they are apt to occur also on the breasts; and in children they are of common occurrence on the legs and shoulders as well as on the hands, arms, and face. The progress of the eruption is attended with more or less itching. The scratching and rubbing that this provokes is the cause of the disease being transplanted from one part of the surface to another.

Herpes tonsurans commences with more or less itching and redness of some part of the scalp. The affected spot is sometimes also slightly swollen; the hair growing from it loses its polish, becomes dull, and is more or less discoloured; it becomes brittle so that it breaks off near to the root. This breaking-off of the affected hairs at the distance of a line or two from the surface of the skin gives the patch an appearance as if it had lately been shaved (hence the name of the disease). If any of these hair-stumps be pressed on by the finger-tip their brittleness and want of elasticity becomes evident by their becoming permanently bent at an acute angle by the pressure, instead of springing back again, or at the most retaining only an almost imperceptible curve, as would be the case with healthy hair-stumps. The epidermis of the patch undergoes a furfuraceous desquamation, the hair-follicles become erect, so that a "goose-skin" appearance is given to the patch, the colour of which becomes changed from a rosy-red to a faint slatish-blue. The hair-stumps as well as the intervening epidermic surface may sometimes be seen to be covered with a fine, white, extremely delicate, fluffy layer like the bloom on a peach. The margins of the patches are abruptly defined; sometimes, especially in lymphatic subjects, successive crops of vesicles, or of pustules, may make their appearance on the shorn patches, which accordingly assume more or less the appearance of patches of Eczema or of Impetigo. There are generally several patches of various size scattered on different parts of the scalp. Sometimes by the coalescence of several spreading patches extensive irregularly-shaped shorn surfaces are formed.

If by means of tweezers it be attempted to pull out one of the hair stumps, it will be found that the stump is so brittle that only a portion of it comes away, leaving the hair root in the skin.

Under the microscope this piece of the stump appears ragged at either of its ends. Instead of breaking with a clean fracture like healthy hair, the broken ends are digitated.

The structure of the hair is greatly altered. Its longitudinal fibres are separated, and the intervals filled with the spores of the Trichophyton. On the surface of the hair are clusters of the same spores. The piece of hair looks something like a bundle of sticks with a number of cherries sticking in clusters to its sides and ends, and stuffed here and there into its interstices.

The spores of the Trichophyton are rounded, have a well-defined outline, and measure about the $\frac{1}{5000}$ inch across.

In the earlier stages of the disease, when the hair has not yet become so brittle as to make it impossible to extract the root, it can be ascertained that the knob of the hair as well as its root-sheath is invaded by the spores of the Trichophyton.

In " Herpes circinatus " the spores are most readily discoverable in the epidermic scurf. By some writers it is believed that the Trichophyton attacks the hairy part of the face of the adult male, whiskers, eyebrows, moustache, and beard, giving rise to the disease known as sycosis, and that it attacks the hair of the axilla, and also the pubic hair of the male and the female, giving rise to a pustular eruption. However it is admitted by those who hold such views that the parasite is very difficult to discover in such cases, and that the eruption is totally different in kind from what is produced by the same parasite on the scalp. These difficulties are got over by assuming that the disease as seen on the scalp is generally in an early stage, whereas on other hairy parts it gets rapidly through its earlier stages, and so comes to present itself commonly in the third stage, and this is an advanced condition that it but rarely attains on the scalp. The difficulty of finding the parasite is accounted for by assuming that it is destroyed by the discharge of the pustular eruption that it provokes. These views, as may have already been guessed, are not shared by the author.

Diagnosis.—"Herpes circinatus" may be mistaken for Pityriasis, Lichen (circumscriptus), Psoriasis (circinata), or Favus. But the patches of *Pityriasis* are not evenly rounded, their redness is of a tawny rather than of a pink or rosy kind, they have not a raised margin, and their centre presents the same appearance as their circumferential part.

In *Lichen circumscriptus*, the patches are less regularly circular, the scales thicker and more harsh, and the centre of the patches as rugous as their outer portion; moreover, the patches spread much more slowly.

In *Psoriasis circinata*, the scales are much larger and thicker; they have a nacreous sheen; the raised ring is much broader as well as more prominent; the reddened skin has a tawny hue; the inclosed area is either sound or exhibits only a tawny yellow, not a pink stain; the rings enlarge far more slowly.

Patches of *Favus* are not very likely to be mistaken for those of "Herpes circinatus," but a microscopic examination of the scales might lead to error unless it be remembered that the sporules of the parasite that produces Favus (the Achorion Schönleinii) are much larger and have more of an oval shape than those of the Trichophyton. The diagnosis between Herpes circinatus and any other eruption will always be assisted by a microscopical examination of the scurf.

"Herpes tonsurans" may be confounded with other diseases of the scalp, for instance with Pityriasis, Psoriasis, Impetigo (granulata), Tinea decalvans or Favus.

But in circumscribed patches, either of *Pityriasis*, *Psoriasis*, or *Impetigo*, occurring on the scalp, the hair of the affected surface, though often more or less thinned, is never broken off uniformly (as in the case of "Herpes tonsurans") at the distance of a few lines from the surface of the skin. The hairs, if pulled at, come away entire, root and all. The patches are not evenly rounded. The hairs do not present the microscopical appearances characteristic of "Herpes tonsurans."

In *Favus* the incrustation, instead of being scanty, slate-powder-coloured, and bran-like, is abundant, thick, and of a bright sulphur-yellow colour; the hairs on being pulled come out readily by their roots—indeed, more readily than healthy hairs—and it may then be seen that their bulbs are much thicker than is natural. A microscopical examination of the hair will clear up any doubt.

Prognosis.—Tinea tonsurans, if it be left to itself, or if it be treated ineffectually, may continue for a very great length of time; for instance, it occasionally happens that cases of two or three years' duration come under notice. After very long continuance the disease is apt to occasion permanent baldness by producing atrophy of the hair-follicles.

In *Tinea decalvans* the patches are completely bald, that is to say, there are no hair stumps; there may be a few colourless downy hairs on the bald patches, but these are almost invisible to the naked eye. The patches are perfectly smooth and polished; they are of a dull white colour.

TREATMENT.—In "Herpes circinatus" but little treatment is required. Washing the patches with a diluted solution of acetic acid (the acidum aceticum, Ph. Br., answers very well), or with a little soft soap, and drying afterwards with a rough towel, is sufficient. Common black writing ink is the popular remedy, and the acetic acid it contains makes it a very efficient one. The *modus operandi* of these remedies is that they dissolve off the semi-adherent scurf, as well as the sup ficial layers of the cuticle, and so detach with the cells the parasi growth that thrives on them. Mercurial ointments of various kin such as the white precipitate ointment, the nitric oxide of mercu ointment, the citrine ointment, and others are also efficient remedie

In "Herpes tonsurans" far more care than is needed for "Her circinatus" is requisite to obtain a good result.

Some authors praise the effect of a solution of sulphurous acid, t destructive influence of that gas on the lower forms of vegetable l being the reason of its employment. The solution is prepared adding vinegar to a solution of hyposulphite of soda, a salt larg manufactured for the use of photographers, and much easier to obta than the sulphite. The resulting solution of sulphurous acid applied by soaking pieces of lint in it, laying them on the affect places and covering them with oiled silk, to prevent the escape of t gas. This ingenious plan, however, is, in the author's experience, from being an efficacious one. In spite of the covering of oil silk t volatile sulphurous acid is dissipated so rapidly, its escape bei favoured by the warmth of the head, that in a surprisingly sh time after the application of the solution the suffocating odour of t gas is no longer perceptible, and very shortly not the least trace of can be smelled. The good done by the solution is, the author believ entirely due to the excess of acetic acid used in its preparation, a to the precipitated sulphur that is formed by the decomposition of t liberated hyposulphurous acid (into sulphurous acid and sulphur).

Epilation, aided by the use of "parasiticides," is advocated ve earnestly by some writers. They all, however, admit that until t disease has begun very obviously to improve epilation is practica impossible, on account of the extreme brittleness of the hairs, l nevertheless advise that the ceremony should be performed regula from the commencement, until later on it becomes really possible extract the roots of the hairs. The object of epilation is to remo the diseased hair roots which are not very accessible to the reach local applications, and thus to render possible the penetration "parasiticide" remedies into the hair follicles. But the object stimulating epilation by pinching off small pieces of the projecti brittle hair stumps is not quite so evident.

In all cases the root of the hair (the part within the follicle) is l diseased than the shaft. It is better, therefore, if epilation be aim at, to improve first, if possible, the state of the stump of the shaft, that it may regain enough of its natural tenacity to bear the strain it necessary to extract the root. This end is best attained by the of some agent which is capable of penetrating the substance of t hair and destroying the parasite. These conditions are found unit in a solution of the hepar sulphuris, a substance procured by fusi together equal parts of sulphur and of dry carbonate of potash, t result of which process is the formation of pentasulphuret of pot sium and hyposulphite of potash. The pentasulphuret of potassi exercises the same solvent effect on the tissue of the hair that is produced by the oxide of potassium, or potash, and carries the dissolved sulphur into every part of the diseased shaft, penetrating even some distance into the root of the hair, as well as into the equally diseased root-sheath. Since the hepar sulphuris is as irritating to the skin as potash, the solution to be used should be of moderate strength.

After the patches have been daily painted with the solution for two or three weeks it will be found that effective epilation can be practised, and then other remedies requiring less care in their use are admissible, for example, sulphur ointment. This should be made of precipitated sulphur, and should contain also a small proportion of the hepar sulphuris, or citrine ointment may be used. A favourite remedy is the nitric oxide of mercury ointment, but it should be made with oxide prepared in the wet way. In all cases during the course of treatment the head should be daily well washed in order to prevent as much as possible the formation of fresh foci of the disease. When epilation becomes practicable it should be performed by means of a broad forceps, having a straight edge. The contingent surfaces of the forceps should be roughened, so that they may "bite" the hair. The sensibility of the surface about to be operated on should first be deadened by means of the ether-spray, and then, the head of the patient being steadied by the hands of an assistant, the patch should be rapidly and thoroughly epilated. When the hair has again grown long enough to afford a good hold to the forceps the process of epilation should be repeated, the ointment being assiduously used in the interim, and so on until recovery has taken place. It is a good plan to persevere in the use of remedies even long after the disease has apparently completely disappeared, since unless every vestige of the trichophyton has been completely destroyed a relapse of the disease is certain to happen.

When the complaint occurs in lymphatic children, some general treatment is advisable. The bowels may require to be regulated, and the diet to be selected, and cod-liver oil and the syrup of the iodide of iron, or steel wine, may be indicated.

Of recent times, the Indian remedy known as Goa-powder (the pith of a leguminous tree found in the province of Bahia in Brazil) has been adopted in this country for the treatment of ringworm. The powder is used in the form of ointment, varying in strength from five to twenty per cent. The remedy is of undoubted service, but care must be taken to avoid the contact of it with the eyes.

TINEA DECALVANS.

Synonyms.—Area; Alopecia circumscripta; Porrigo decalvans; Pelade; Phyto-alopecia; Vitiligo; Teigne pelade; Alopecia areata; the smooth Ringworm.

Definition.—A contagious disease produced by a vegetable parasite which, attacking the hair, leads to the formation of circumscribed, pale, smooth, bald patches.

Causes.—(Predisposing.)—Childhood; constitutional syphilis. (Exciting.)—The presence of the *Microsporon Audouini.*

Description.—The disease commences (usually on some part of the hairy scalp) with slight itching. The itching is confined to a limited spot, the hair growing from which may on examination be found to have lost its natural gloss, as well as its natural firmness of attachment to the skin, since on pulling at it it readily comes off. On examining, by touch, the corresponding patch of skin, it may be ascertained that it is slightly indurated. Very soon the loosened hair falls off, and it may be seen that the disease occupies a small well-defined area, which is completely denuded of hair. On a cursory view the most striking peculiarities of the disease at this stage are the perfect baldness of the patch, which is smoother than the most closely shaven beard, the absence of any other notable change in the affected skin, and the abrupt manner in which the baldness ceases at the margin of the patch, the hair immediately surrounding which is as thickly set and as long as on any other part of the scalp.

On examining more closely, however, it may be found that the skin corresponding to the bald patch is slightly thickened and indurated, is somewhat paler than the surrounding skin, and has something of a polished appearance, and furthermore, that the hair immediately around the patch has lost its gloss, and may be easily pulled out. The baldness, too, is not so absolute as it at first sight seems, for the hair that has been lost becomes very speedily replaced by a scanty crop of short, light-coloured, thin, silky filaments, which resemble the fibres of cotton or rather of fine wool, and may require a lens for their detection, while towards the margin of the patch short, thick, club-shaped hair-stumps make their appearance. These stunted hairs vary from the $\frac{1}{16}$ to the $\frac{1}{8}$ of an inch in length, and are mostly of the same colour and thickness as the healthy hairs, except at their free extremity, which is always of a much darker colour as well as much thicker than the lower portion of the stump.

If a hair be taken from any part of the skin where the disease is in active progress, *e.g.*, from the edge of a bald patch which is spreading, it will probably exhibit (under the microscope) traces of the Microsporon Audouini. The hair should be moistened with a dilute solution of caustic potash and viewed through a "quarter" object-glass. The spores of the Microsporon Audouini, which are smaller and less numerous than those either of the Tricophyton tonsurans or of the Achorion Schönleinii, may be seen scattered in the form of minute spherical granules in the substance and on the surface of the diseased hairs.

The disease may spread from only one, or from as many as ten, or even twenty independent centres. As a rule, when it first comes under observation, there are at least three or four separate patches, varying in size from that of a threepenny-piece to that of a crown.

Varieties.—The disease may assume either of two phases. In the one variety (for which the author proposes the name Alopecia nummulata) the patches increase but slowly, so that they may be several months attaining a diameter of one or two inches, and their form is evenly rounded, being either oval or circular.

In the other (much rarer) kind, which may be distinguished as Alopecia serpiginosa, the progress of the disease is more rapid and its extension takes place in a less regular manner; the bald places speedily spread so as to form large irregularly shaped patches with sinuous margins.

Occasionally the two varieties may co-exist in the same individual. The nummulated variety may sometimes, by the coalescence of two or three patches, simulate in some degree the serpiginous form; but even when this happens the margin of the compound patch will exhibit only three or two regular curves corresponding to the outlines of the patches which compose it, and may be readily distinguished from the regular border of the serpiginous variety. In the former case, too, the patient's account of himself will prove that the disease has remained for some time in the condition of separate rounded patches before the irregular patch was produced. The distinction, which may perhaps appear of but small importance, is of value in determining the prognosis which, with reference to the chances of permanent baldness, is much less favourable in the serpiginous than in the nummulated variety. Indeed, in the former the progress of the disease is sometimes so rapid and extensive that in a few months from its first appearance the patient may be left for the rest of his life without a single hair on any part of his body.

Tinea decalvans, when left to itself, may terminate either in (spontaneous) recovery, or in incurable baldness; the former result, which is comparatively rare, occurs more frequently in the nummulated than in the serpiginous variety. The approach of recovery may be recognised by an arrest of the extension of the disease, by the cottony fibres gradually acquiring more and more the characters of healthy hair, that is to say, becoming coarser, longer, and of a darker colour, and by the diseased skin regaining its natural colour and losing its induration.

When permanent baldness has been produced the induration of the skin disappears, and it regains almost if not completely its natural colour, but instead of remaining at its original thickness and consistence it becomes thinned and atrophied. On the scalp and other parts where the hair is naturally coarse and thickly set, this alteration becomes very manifest. In this condition of the skin the cottony fibres become very fine and scanty or may be altogether wanting.

In either of its forms the disease is commoner with children than with adults. In the latter it often coincides with recent constitutional syphilis, as if the modification of the system produced by the syphilitic virus offered a favourable soil for the development of the disease. It will be understood that what is meant here is the veritable Tinea decalvans, as distinguished either from general (syphilitic) thinning of the hair, or the bald cicatricial patches that are left by some syphilitic eruptions.

Diagnosis.—Tinea decalvans may be mistaken for Vitiligo, Tinea tonsurans, Tinea favosa, Senile Alopecia, Syphilitic Alopecia, or the temporary loss of hair that is often consequent on various acute illnesses.

But in *Vitiligo,* although the hair be discoloured, it retains its usual length, as well as its natural degree of coarseness. There is no induration of the skin, and the paleness of the patch itself is made up for by the unnaturally deep colour of the skin in its immediate neighbourhood, and furthermore the disease does not exhibit that marked predilection for the scalp which is noticeable in Tinea decalvans.

In *Tinea tonsurans* the skin is darker than natural, having a greyish tinge; the surface, which is scurfy, has a "goose-skin" appearance, the hairs are not absent, but merely broken off close to the skin.

In *Tinea favosa* the presence of small yellow cup-shaped incrustations, or of the larger white powdery crusts, and the inflamed condition of the skin itself, will usually suffice to avoid error.

In *Senile and* in *Syphilitic Alopecia* the baldness is diffused, not circumscribed; there is a general thinning of the hair, and no formation of circumscribed bald patches. The same remarks apply to the baldness produced by pregnancy, or by acute general disease.

The bald patches which are caused by cicatrices, resulting whether from injury or disease, may be distinguished by the cicatricial character of the integument, and by the history of the case.

Prognosis.—In the *nummulated* variety it is generally favourable as regards the prospect of ultimate recovery, although if the disease be of long standing, a period of several months may sometimes be required to restore the hair to its original condition. In the *serpiginous* variety the prognosis will be doubtful; indeed the author has met with cases in which, notwithstanding that the patient had had from the first the advantage of eminent advice, complete and permanent baldness had ensued. In either variety the probability of speedy recovery will be less in proportion to the previous duration of the disease, the size of the patches, the fineness and scantiness of the cottony filaments, and the atrophy of the scalp.

Treatment.—The administration of arsenic internally has been recommended for this disease, but the author's experience of it is not at all such as to induce him to recommend it here, as of any, even the slightest, use. By many the effect of steel has been praised, but although of unquestionable service in the temporary baldness following acute general disease, it is quite incompetent *per se* to arrest the progress of Tinea decalvans, and it is doubtful whether its employment is of any advantage. This much, at all events, may be said, that the usual indication for steel, viz., pallor, is as often absent as present in this affection.

The local applications that are in good repute for the healing of bald patches, are:

Ointment of tar (ʒij. to the oz.), the Unguentum Cantharidis, the Unguentum Creasoti. Ointment of the Huile de Cade (ʒj. to the oz.). The Linimentum crinale (which is composed of Cantharidine gr. j. Spirit. Rectif. ℥iij. Ol. Ricini ℥j.). The Cantharidine tissue, or the Capsicum tissue (sold under the name of Sinapine tissue). Alcoholic solutions of the various essential oils, in the proportion of ʒss. of the oil to ℥j. of rectified spirit. Ointment of the Sulphur præcipitatum (ʒij. to the oz.). The Unguentum Hydrargyri mitius of the Dublin Pharmacopœia. The Unguentum Hydrargyri Iodidi Rubri, diluted with three times its bulk of simple ointment. An ointment of Turbith-mineral (gr. xv. to the oz.). An aqueous solution of corrosive sublimate (gr. ij. to the oz.), and others.

All that have been mentioned, it will be observed, are either "parasiticide" or "stimulant," while many of them possess both of these properties. There is no doubt but that stimulation is an essential part of the process of cure, and, according to the author's experience, it is better that the stimulation should be slight and continuous than violent and intermitting.

SYCOSIS.

Synonyms.—Chin-welk; Herpes pustuleuse; Mentagre; Felgwazen; Phyma; Mentagra; Tinea sycosa.

Definition.—Sycosis is a disease of the hairy part of the face, caused by the growth of the *Microsporon mentagrophytes* in the root-sheaths of the hair, and characterised by considerable cutaneous and subcutaneous inflammation, and the development of pustules and nodular swellings.

Causes.—(Exciting.)—Contagion. (Predisposing.)—Adult or advanced age and the male sex.

Description.—The eruption is confined to the hair-covered portions of the face. It is consequently only met with in adult males, or (very rarely) in such adult females as are provided with more or less of a beard. It exhibits a special predilection for the *chin*, whence its names Mentagra and Chin-welk. The disease commences with a sensation of heat and tingling of the affected surface, which soon becomes reddened, scurfy, tender, and somewhat swollen. Soon pustules and tubercles of a peculiar kind are developed on the inflamed patch.

The pustules are very large, and are purulent only at their summits, the indurated acuminated base, which may attain the size of a nut, forming the greater part of the pustule. The pustules are sometimes isolated, but are generally arranged in clusters.

The tubercles, which may attain a size equal to that of the pustules, occur in circumscribed groups, those of the same group running int one another, and forming a raised nodulated patch. The surface this patch soon becomes excoriated, and secretes a muco-purulen fluid, which concretes into a thin dirty-grey scab. On detaching an removing this scab, a moist, glistening, coarsely-nodulated red surfac is disclosed, looking something like the inner part of a ripe green fi (hence the name Sycosis). The surface of a ripe mulberry is perhap a better simile.

These circumscribed, raised, nodulated, moist scab-covered patche (*fungous ulcers* as they are sometimes termed) occurring about th chin, are very distinctive of the disease.

The pustules, or the tubercles, attain their full development withi the space of ten days or a fortnight.

As might be supposed, so much acute inflammatory swelling of th chin is attended with considerable discomfort, and produces enlargement, and sometimes suppuration, of the submaxillary glands.

The hair of the affected surface soon becomes loosened, and falls, o is only maintained *in situ* by its roots becoming entangled in th crusts. When the crust is removed nearly all the hairs that it ha entangled come with it.

Sycosis is in this country an extremely rare complaint, rarer eve than Favus. On the Continent, however, it is not rare.

Prognosis.—If submitted to suitable treatment Sycosis may b recovered from, but if left to itself it is likely after a long continuanc to end in permanent baldness.

Diagnosis.—Sycosis may be mistaken for impetigo sycosiformi for a vegetative or a tubercular syphilide, or for epithelial cancer.

Sycosiform impetigo, however, is a more superficial eruption. I pustules are never so large, nor are they ever accompanied with s much infiltration of the skin and cellular tissue as those of Sycosi It presents, so to speak, much less of a malignant appearance tha Sycosis. There are no "fungoid ulcerations" in sycosiform impetig It exhibits no special predilection for the chin. On the contrary it commoner on the upper lip than elsewhere, and is very often limite to the hairy parts of the cheeks. On detaching the crusts of sycos form impetigo, the entangled hairs do not come away with the crust but remain firmly attached to the inflamed skin, nor is any notab baldness of the affected surface produced by the eruption, unless th disease be of very long (several years) standing. Sycosis is contagiou impetigo not so. A microscopic examination of the hair growing fro the inflamed skin may, in cases of doubt, be called in to aid the dia nosis. In arriving at a decision it should always be remembered th in this country an eruption of the chin is far more likely to be sycos form impetigo than Sycosis.

Acne (indurata) may be readily distinguished from Sycosis by i not being confined to the hairy part of the face, by its tubercles bein of much slower growth, by their containing a sebaceous core, and b the absence of "fungous ulcers."

The flat vegetative syphilide, or mucous tubercle, is apt to be mistaken for Sycosis because it is not uncommonly developed in the neighbourhood of the mouth, and presents a raised raw surface covered with a crust. But the surface of the mucous tubercle is paler and is much smoother than that of the ruddy, coarsely-nodulated, fungous ulcer of Sycosis. Its margin often overlaps the constricted base—a condition never present in Sycosis. It is commoner at the angles of the mouth and on the cheeks than on the chin.

The tubercular syphilide (the clustered non-ulcerating variety of which may be thought to have some resemblance to Sycosis) is far more slowly developed. Its tubercules either do not form a raw surface, becoming gradually re-absorbed without breach of surface, or if a raw surface be formed the crusts covering it are thick and dark-coloured, and conceal, not a reddened nodulated fungous surface, but abruptly-limited sunken ulcers, with flat ash-grey floors. The tubercular syphilide is always accompanied by other symptoms of constitutional syphilis.

Epithelial cancer is a disease of far slower progress than Sycosis. It presents, it is true, like Sycosis, an elevated raw surface, but the circumferential is decidedly more elevated than the central part, and the surface is lobulated rather than nodular. The disease, when it occurs in the neighbourhood of the chin, affects usually the lower lip.

Treatment.—The treatment of Sycosis is to be conducted on the same principles as that of Tinea favosa.

CHLOASMA.

Synonyms.—Liver-spots; Taches hépatiques; Crasses parasitaires; Danne hépatique; Leberflechte; Pityriasis versicolor; Maculæ hepaticæ.

Definitions.—A contagious disease produced by a vegetable parasite (the *Microsporon furfur*) which, attacking the epidermis, leads to the formation of more or less extensive, tawny-yellow, scurfy patches.

Causes.—(Predisposing.)—Adult age; the tubercular diathesis; pregnancy. (Exciting).—The presence of the *Microsporon furfur.*

Chloasma scarcely ever occurs in children. Tuberculosis appears to be a favouring cause. The disease is apt to occur in pregnant women, disappearing often shortly after delivery. It has been supposed that wearing flannel is a cause of the disease; the great argument in support of this theory is the fact that the eruption ordinarily is limited to the parts usually covered by a flannel under-garment, viz., to the trunk and the upper parts of the arms and thighs.

The author has often met with Chloasma occurring in those who do not wear flannel underclothing, and in them the disease has been as strictly confined to its usual situations, as it is in those who wear flannel all the year round.

Description.—Chloasma, or as it is often called, Pityriasis versico presents the appearance of variously-shaped stains of the skin, wh may be of a yellow, a brown, a reddish, or even a black colour. Th discoloured patches may be small, rounded, and distinct from another, or larger and coalesced so as to form fantastic patterns, outlines of which, although sinuous and irregular, are usually defined. In other cases one large uniform patch may cover nea half the body. The surface of the patches is slightly raised above level of the skin, and exhibits a mealy desquamation. This scurfin though it is much less conspicuous than that of Pityriasis pro is important as a means of diagnosis. According to the auth observation it is always most perceptible at the margin of the dise When not evident over the general surface of a patch, it may rendered so by scraping the skin lightly with the back of a penkn On detaching the diseased epidermis the skin beneath is seen to *reddened* as well as swollen. The epidermis itself is yellow. disease commences as small scattered spots of about the size of a pi head; these slowly spread till they meet and coalesce with one anot

Chloasma is seen most frequently on the anterior surface of neck and of the upper part of the chest, and on the upper part of back and arms; it is common, too, on the pubic region and the in half of either groin. The author has remarked that it rarely aff the hollow of the axilla, or the lateral surfaces of the chest or ab men, even when most extensively spread over the anterior posterior surfaces of the trunk.

Chloasma is more commonly a disease of adult age. It is seen of in the phthisical. It follows a chronic course. It depends essentia on the presence of the *Microsporon furfur* in the substance of epidermis. If a portion of the epidermis scraped off from diseased surface be moistened with dilute acetic acid or a solution potash and placed under the microscope, the cryptogam will be seen consist of (*a*) clusters of cells (the sporules), and (*b*) ramified tubu (the mycelium). The various lines of different patches of Chloasma the same individual depend on the varying quantity in which Microsporon furfur is present at different parts of the surface. itching that attends Chloasma is usually slight, but it is occasiona severe.

Chloasma is apt to occur in pregnant women, disappearing of shortly after delivery; singularly enough in such cases the face is favourite locality of the disease. This Chloasma gravidarum must distinguished from the Ephelis of the pregnant, which it mu resembles in general aspect, but which is produced by an alteration the pigmentary matter of the epidermis, and is attended neither w itching nor with desquamation.

Prognosis.—If untreated the disease continues indefinitely. Bu yields readily to suitable treatment. It is, however, very apt to retu after a time. When it occurs in pregnant females it may be expec to disappear shortly after delivery.

Diagnosis.—In no other eruption are the characteristic spores and mycelium of the Microsporon furfur to be found in the epidermis. Chloasma may, however, be mistaken for Ephelis, for Pityriasis, or for Vitiligo. But in *Ephelis* there is no itching and no scurfiness, in *Pityriasis*, although there is both itching and scurfiness, the scales are white. In *Vitiligo* the lighter patches inclosed by the darker stains are unnaturally white, whereas in Chloasma they are of the natural colour. There is no scurfiness in Vitiligo.

Treatment.—Lotions of sulphurous acid are recommended in the treatment of this disease, but sulphurous acid is far too volatile an agent to be of much service. When exhibited in this shape lotions of dilute acetic acid are of service, they soften and detach the diseased epidermis and remove with it the parasite. Weak lotions of caustic potash or strong solutions of the subcarbonate of potash have a similar effect. Dilute lotions, or still better, weak baths, of corrosive sublimate are very efficient remedies. So are sulphur baths.

A very efficacious mode of treatment is the following:—The patient first thoroughly soaps the affected skin; after this he takes a warm bath, and as soon as he has dried himself, scrubs the discoloured surface with a flesh brush; he then rubs well in, over every patch of the disease, a weak mercurial ointment. This process is repeated every second day. This plan, which the author has employed pretty extensively, has the following advantages. By the first operation all the semi-detached scurf and much of the parasite is removed. By the second the remaining epidermis is thoroughly softened, so that by the third step the more superficial portion of it, with the parasite matter contained therein, is readily detached, and thus the more deeply seated portions of the fungus are laid bare to the immediate contact of the remedy. By this process patches that have existed for many years may be removed completely in about a week's time. An ointment of sublimed sulphur has been recommended, but the precipitated sulphur is a much more active remedy than the sublimed sulphur. If sulphur ointment be used, its efficacy is much enhanced by the addition of a small quantity of the hepar sulphuris.

Chrysophanic acid ointment has of late been used with considerable success in the treatment of Chloasma. Twenty per cent. ointment may be used for the purpose, special care being taken to keep the remedy away from the eyes.

SECTION XII.

FOREIGN DISEASES OF THE SKIN.

Of the diseases of the skin that are foreign to this country shall describe the following:—Elephantiasis Græcorum; Elephanti Arabum; Frambœsia; Pellagra; and Malum Alepporum.

ELEPHANTIASIS GRÆCORUM.

Synonyms.—Mal de S. Lazare; Mal rouge de Cayenne; Maladie Jérusalem; Elephantenaussatz; Der knollige Aussatz; Spédalsk (of the Norwegians); Leontiasis; Satyriasis; Lepra of the Ara Elephantiasis tuberculata; Lepra tuberculosa; Tsarath (of Hebrews); Dsjuddam or Jusam (of the Arabians).

Definition.—Elephantiasis Græcorum is an extremely chronic gene disease which is characterized at first by the development of taw stains on the skin, the sensibility of which is either (most common diminished or (less frequently) increased subsequently by yellowi brown tubercles, and finally by ulcers which often extend dee into the subjacent tissues.

As the disease progresses it is attended with considerable debil and mental dejection, and in most cases terminates fatally from ulce tion of the bowels and colliquative diarrhœa.

Causes.—Greek Elephantiasis was formerly thought to be c tagious. It has, however, long been satisfactorily ascertained that is not so.

The predisposing causes of the disease are *hereditary transmiss* and the *influence of special climatic conditions;* it is consequen endemic in certain localities. Climate appears to be a more essen cause than hereditary predisposition.

The exciting causes are uncleanliness, unhealthy habitation, inte perance, an unwholesome diet, debauchery, great fatigue, nerv affections.

The disease is commoner with men than with women; it is genera developed before puberty.

Description.—Generally, the first symptom of the disease is appearance of small circumscribed stains, which, though usually o tawny, are sometimes of a purple colour. In negroes the stains sometimes of a darker, sometimes of a lighter colour than the affected skin.

Occasionally, however, the appearance of the stains is preceded several weeks or even months by general symptoms: languor and feeling of weight in the limbs; less commonly by feverishness.

The stains appear at first on the face, legs, and forearms. On face (where they are often for some time confined to a limited spac the ears or the nose) they are accompanied with considerable sw ing of the subjacent cellular tissue.

The sensibility of the skin corresponding to the stains is in all cases considerably altered; generally the sensibility is diminished, sometimes so much so that they may be pricked with a needle without the patient being conscious of it. But in some instances the sensibility is increased so that the least touch occasions the most vivid pain, producing a sensation which has been compared to that caused by a blow on the nerve at the elbow.

The disease may continue in this stage for several years, the stains after a time remaining stationary or continuing constantly to invade fresh regions.

At length small tumours take the place of the stains: they are of irregular shape and various size, ranging from the size of a pea to that of a walnut; their colour is yellowish-brown or (sometimes) dusky-red; they are soft and shining. The face is the situation where they increase most rapidly and become most developed, so that the countenance at length presents a revolting and even hideous appearance—it becomes studded with irregular knobbed elevations, separated here and there by deep furrows—the skin is much hypertrophied and the subcutaneous cellular tissue considerably swollen—the brows are overhanging—the lips, ears, and nose, enormously thickened—the eyebrows, eyelashes, and beard fall off, and the whole face is of a tawny brown or dusky hue—its dreadful deformity is rendered the more disgusting by a greasy film which lubricates the tubercles and gives the skin a shining appearance. From this hideous deformity of the face the disease has acquired its names. "Leontiasis" "Elephantiasis" "Satyriasis." The last of these names, which refers only to the disfigurement of the face, has given rise to the erroneous supposition that the sexual propensities are morbidly increased in this disease.

On the limbs the disease presents the same characters, but in a milder degree than on the face.

In the tubercular stage the alteration in the sensibility of the affected skin is usually more marked than in the antecedent macular phase.

But it is not only on the skin that the tubercles of Greek Elephantiasis are developed. They invade also the conjunctiva (ocular as well as palpebral) and the mucous membrane of the mouth, nose, pharynx, and larynx, so that sight and hearing are affected, smell and taste become greatly impaired, the voice becomes husky or extinguished, respiration more and more difficult, and the patient, weak and dispirited, loses all power of exerting himself.

The disease continues in the tuberculous stage usually for several years.

At length the tubercles inflame and ulcerate. Sometimes the ulcers are fungous and superficial, and produce and renew moist dark incrustations, which from time to time get detached. In other cases they eat deeply into the subjacent tissues. As the disease advances the internal organs become affected; the trachæa and the ramificatio of the bronchi, the œsophagus and the intestines, become the seat albuminoid deposits, similar to those of which the tubercular elev tions of the skin are mainly composed. In these situations, as on t skin, the deposit at length undergoes softening, and so ulcers are pr duced, and the disease terminates fatally with enteritis and colliqu tive diarrhœa.

The above description refers to what has been termed the tuberc lated variety of the disease (Elephantiasis or Lepra tuberculata Sometimes, however, the stains, in place of being succeeded tubercles, are replaced by bullæ resembling those of chronic pemph gus; these, after breaking, leave sores. This latter variety is calle *par excellence* E. or L. anæsthetica. The ulcers which, as in the oth variety, often extend deeply, are covered with white scabs. The ski at first extremely sensitive, gradually becomes parchment-like a loses its sensibility, so that the greater portion of the surface m become affected with anæsthesia. The limbs become stiff and inflexib and the muscles atrophied. Ulcers encircle the fingers or toes, whi become separated and drop off. Violent pains are felt in the limb and the disease ends fatally with diarrhœa and sometimes tetan spasms.

Sometimes the two varieties coincide in the same individual, that the face may be affected with the tuberculated and the lim with the bullous eruption.

The course of the disease is usually spread over a long period—t or even twenty years.

Respecting the nature of the disease it has been attributed Danielssen to a dyscrasy consisting in an excess of albumen a fibrin in the blood.

Although differing widely in many particulars from syphil scrofula, and tuberculosis, it resembles them in so far as it is t result of a special diathesis, which is manifested by a depravati of the general health and the deposition at various parts of t body of a lowly organised material which after a time softe and breaks down. The lesions of sensibility, which are so char teristic of this disease, are attributed by some to the depositi of albuminous material on the surface and in the substance of t nervous centres, and by others to a similar deposit in the sheaths the nerves.

Elephantiasis Græcorum is endemic in all tropical latitudes, a also in some high northern latitudes, viz., in some parts of Norw and Sweden. In this country it is occasionally met with in foreign who are natives of countries where the disease is prevalent, or Englishmen who have long resided in such countries. Several ce turies ago it was endemic in England and in many parts of Euro whence it has since disappeared. Cases have occurred in persons English parentage but born in a country where the disease preva but who nevertheless have been quite free from the disease until even a year or two after their arrival in England, so that the disease has come to be erroneously attributed by the patient to the influence of the English climate.

Diagnosis.—When the eruption of Elephantiasis Græcorum is in the macular stage it may be mistaken for Erythema, for Pityriasis versicolor, or for a Syphilide, but the peculiar alteration in the sensibility of the discoloured skin will at once serve to distinguish this disease from any other; later on it is almost impossible to confound it with any other eruption.

Prognosis.—This is always a most serious disease, and it terminates in most cases fatally. In some instances, however, it has been recovered from. Such an issue may be hoped for when the patient is young and vigorous and the disease has not already made much progress.

Treatment.—Many specifics for this disease have been brought forward but none of them has acquired anything like a lasting reputation, and it is more than doubtful whether any of them can be truly said to exercise control over the progress of the complaint.

The avoidance of such causes (indicated above) as are known to favour the progress of the disease. The internal administration of cod-liver oil, of arsenic, of iodide of potassium or iron, and the employment of stimulating applications to the stains or tubercles, and of caustics to the ulcers, are the best measures that can be adopted. When enteritis or diarrhœa have supervened the diet should be carefully regulated and the use of opium will be called for.

ELEPHANTIASIS ARABUM.

Synonyms.—Barbadoes-leg; Egyptian sarcocele; Lèpre tuberculeuse éléphantine; Hernie charnue; Elephas pachydermia; Bucnemia; Hypersarcosis; Febris erysipelatosa; Dal. fil (of the Arabians).

Definition.—Elephantiasis Arabum is a disease of the lymphatic vessels. It is characterised at first by a series of attacks of acute inflammation of the lymphatic vessels of some limited region, accompanied sometimes with erysipelatous inflammation of the skin of that region. These attacks succeed one another after distant intervals, and lead finally to a considerably swollen and indurated condition of the part affected.

With the Elephantiasis of the Greeks this disease has nothing in common but the name.

Causes.—The etiology of this disease is very obscure. Climate has undoubtedly a considerable influence in its production, since it is almost peculiar to tropical countries. It is especially common in certain parts; for example, Rio Janeiro and Barbadoes. From the latter place it derives its common English name. It has been attributed to the great diurnal variations of temperature that are peculiar to tropical climates—to the contamination of the atmosphere with the exhalations from decomposing organic matter. It may occur at all ages but is commoner with adults than with children. It affects both sexes indifferently. It is not an hereditary nor is it a contagious disease.

Description.—The disease commences with general febrile disturbance and acute pain in the part about to be be affected, the inflamed lymphatics of which soon form tender swollen knotted cords. When the complaint occurs in the leg a hard knotted cord runs up from the ankle or knee as far as the groin, where it ends in the swollen glands; at the same time the skin of the affected part is commonly affected with erysipelatous inflammation.

The attack is attended often with vomiting and headache, and sometimes even with delirium, but in a few days the patient is well again, the only relic of the attack being a slight swelling of the affected part. After an interval, which varies from one to many months, the attack is renewed, and as the paroxysms are frequently repeated, and each time leave the affected part more and more swollen, the enormous solid swelling which distinguishes the disease is at last produced. It is not, however, until the disease has lasted many years that the swelling acquires any very considerable development.

There are many variations in the appearance of the swollen part: sometimes the swelling is uniform and rounded, sometimes in successive portions separated from each other by deep folds. On the leg these folds may cease at the foot, which retains its natural appearance, or the foot may be involved in the swelling; in the latter case the limb resembles the leg of an elephant—the swelling, however, never involves the sole of the foot, nor when the arm is affected does it ever extend to the palm of the hand.

In some cases the skin of the swollen part is thin, smooth, and polished, so that if the swelling be uniform it presents the same appearance as in ordinary œdema, but there is this difference, tha in Elephantiasis the surface is harder and firmer, it does no readily pit cn pressure. In other cases, especially after a paroxysm the swollen surface may present a papular or a vesicular eruption. Sometimes the skin may be disfigured by a varicose state of the smalle veins, sometimes it acquires a scaly condition as in ichthyosis sometimes it becomes covered with warty growths, which have been compared to syphilitic warts, and then deep cracks may be formed which become covered with crusts which, from time to time, become detached and are renewed. In such cases portions of the skin may ulcerate, or even large sloughs may be formed. Occasionally the lymphatic glands suppurate. When death results from the disease the patient sinks gradually, and at length dies of hectic fever.

The disease may occupy almost any part of the body. Thus it has been noted on the scalp, the ears, the neck, the trunk, the scrotum, the penis, the mammæ, the vulva, and the limbs. Its most favourite situations are the limbs, especially the lower limbs and the scrotum. When it occurs on the leg (its commonest situation), it is known as Barbadoes-leg. The next most frequent seat of the disease is the scrotum; when it affects this part it is known as Egyptian sarcocele; in this situation the swelling is often complicated by an abundant sebaceous exudation from the skin.

The scrotum affected with Arabian Elephantiasis sometimes attains enormous dimensions; thus it has been known to measure four feet in circumference, and to weigh as much as from seventy to eighty pounds.

The swelling of Arabian Elephantiasis is produced by a plastic exudation in the meshes of the subcutaneous areolar tissue which becomes developed into dense areolar or fibrous tissue. Sometimes the exudation is deposited also in the meshes of the intermuscular areolar tissue and the muscles become atrophied and undergo fatty degeneration.

Diagnosis.—By a reference to what has been said in our description of Elephantiasis Græcorum it will be seen that Elephantiasis Arabum can scarcely be mistaken for it.

From œdema the swelling of Elephantiasis Arabum may be readily distinguished by its hardness, by its abrupt margin, and by the history of a series of erysipelatous attacks having preceded it. When, however, the skin has undergone the alterations we have described as common in Elephantiasis Arabum, the swelling no longer presents the appearance of mere œdematous infiltration.

Prognosis.—The disease has an indefinite duration. In its early stage a favourable termination may sometimes be hoped for, but when of long standing, Arabum Elephantiasis may be regarded as incurable.

Treatment.—In its early condition, when the disease consists of a series of acute inflammatory attacks, these should be combated by a restricted diet, the application of leeches around the inflamed part, and continued poulticing.

In the later acute attacks dry cupping is useful when permanent swelling has become established. Frictions with mercurial ointment will often produce decided benefit. Iodine or iodide of potassium ointment have been recommended, so have blisters and cauterisation of the skin, but any measures that have the effect of inflaming or irritating the skin must be regarded as of doubtful use. Wet cupping has been recommended.

Well-regulated pressure constantly kept up on the swollen part is often of considerable service. The effect of the vapour-douche has been praised.

The internal treatment best calculated to reduce the swelling of Elephantiasis is the prolonged systematic administration of mild purgatives, and of moderate doses of the iodide of potassium.

Amputation is occasionally practised for the relief of Elephantias of the leg, but except in cases where the disease is very far advanc it is not to be recommended; the removal of the whole of t diseased part does not necessarily effect the cure of the disease, sin after amputation it has been known to return in other remote par of the body.

Ligature of the main artery of the affected limb (for instance of t femoral when the leg is affected) is apparently a preferable procedu and has occasionally been followed by good results.

FRAMBŒSIA.

Synomyns.—Yaws; Sihens or Sivvens; Pian; Schwammförmig Lepra fungifera; Mycosis.

Definition.—The Yaws is a contagious disease, appearing once on during life, running a definite but chronic course, and characteris by the eruption of a number of raspberry-like tumours on certa parts of the skin.

Causes.—The disease is indigenous in Central Africa (where it known as the Yaws), hence it has been conveyed to the West Indi (where it is called Pian). It is known also in the northern parts the British Islands under the name of Sivvens. It is commo with children than with adults, and with negroes than with wh persons. It is propagated by contagion of the matter discharged fr the eruption; it is therefore inoculable. It is believed that the dise is extensively spread by the medium of flies that have lit on the so of affected persons.

Description.—The period of incubation of the disease is about t months. It appears first as small red points like flea-bites, th soon rise into pimples, which extend till they attain on the aver half an inch in diameter. As these tubercles enlarge, their surf becomes covered with a scab. Beneath this seat a fungus grow consisting of florid prominent granulations springs up. From this fu gous growth the disease derives it name Frambœsia (*framboise* raspberry). Two or three months elapse before the red point atta the raspberry-like condition. While the first crop of tubercles attaining maturity, a second crop will sometimes make its appearan After the eruption has continued some time it usually happens t one of the tubercles will increase considerably in size, so as become much larger than any of the others;—it may attain a diame of a couple of inches. This, which is called the Mamma-pian, Mother-yaw, becomes corroded and assumes the form of a depres ulcer, while the other yaws continue raised; it generally leaves a s behind it.

After remaining stationary for some time the raspberry-like tumo become atrophied, gradually sink to the level of the skin and disa pear, leaving sometimes, but not usually, a faint scar.

When the tumours are very numerous they are generally smaller than when they are few. The course of the disease is very slow, extending in the case of adults generally over a year, or even a year and several months; in children its duration may be stated at seven or eight months.

The eruption may affect any portion of the skin, but the parts chiefly affected are the scalp, face, axillæ, groins, and the neighbourhood of the genitals and anus.

The little tumours cause no pain except when they occupy the palms or soles; the unyielding epidermis here binding them down in these situations; they are called crab-yaws.

The yaws are not necessarily attended with any constitutional disturbance. Sometimes at their first appearance there is slight feverishness, and sometimes their decline is attended with more or less general debility.

Diagnosis.—Frambœsia cannot very well be mistaken for any other disease.

Prognosis.—In cases of this disease it may always be anticipated that after a duration of several months the disease will disappear without any serious result.

Treatment.—There are no means of cutting short the disease. It must be allowed, within certain limits, to take its own course.

So long as fresh tubercles continue to appear the treatment should be limited to such measures as cleanliness and a nutritious diet.

When the disease has begun to decline, the usual practice is to administer sudorifics, such as the compound decoction of sarsaparilla, antimonial preparations or sulphur—and such tonics as quinine, iron, or the mineral acids.

As local applications to the tubercles themselves, mildly stimulant applications, such as ointment of the oxide or of the subiodide of mercury may be used. If cauterization be required, the arsenical paste of Frère Côme or the acid nitrate of mercury are the favourite applications.

PELLAGRA.

Synonyms.—Elephantiasis italica; Dermatagria; Pellarina; Malattia di miseria; Mal. rosso; Mal de misère.

Definition.—Pellagra is a chronic periodic disease peculiar to Northern Italy, Southern France, and a part of Spain, characterised by red or chocolate-coloured desquamating patches on the exposed parts of the skin, and disorder of the digestive organs and of the nervous system.

Causation.—Pellagra has been attributed to "an endemic and special influence of the soil." It is often hereditary. It is a little more common with women than men. It generally affects middle-aged persons. It is almost confined to the very poorest classes, hence its name "Mal de misère." Unhealthy miasmata, excessive bodily labour, insufficient or unwholesome food, crowded and ill-ventilated dwellings, mental anxiety and distress, have been enumerated as predisposing causes of this disease. It has been attributed to the use of maize as a common article of diet, and especially to unripened maize, which is often attacked by the Sporiosorium maïdis to which fungus a special influence has been ascribed. Insolation is believed to be an exciting cause. The disease is not contagious.

Description.—Pellagra (pellis ægra, diseased skin) is a general disease which begins (usually in the spring), by malaise, a feeling of depression, both mental and bodily, an anæmic aspect and various symptoms of digestive derangement.

As the spring advances and warm weather commences an eruption appears, which affects especially those parts of the body that are left uncovered, and so exposed to the direct rays of the sun, viz., the face, the neck, and chest, the fore-arms, and the backs of the hands, the lower parts of the legs and the upper surfaces of the feet.

The eruption may consist either of patches of dark erythematous redness (without swelling) of the skin, with generally slight scurfiness, or of chocolate-coloured stains accompanied with a scaly condition of the cuticle resembling psoriasis or ichthyosis. Sometimes the eruption is of an erysipelatous character with vesicles or blebs, and a burning pain, which end in exfoliation of the cuticle.

During the progress of the eruption the general symptoms continue. The appetite is either wanting or excessive, the bowels constipated or loose with offensive discharges, the lips and mouth are livid and sometimes ulcerated.

Towards the beginning of autumn the skin affection disappears and the general symptoms become less marked. The patient is in every way better till the return of spring, when he becomes worse than before and the eruption reappears. The general cachexia deepens and the disorder of the digestive system is aggravated. More than this, disease of the nervous system is now superadded, pains in the back and limbs, cramps in the legs and other spasmodic symptoms, great loss of muscular power, amounting almost to palsy, especially in the lower limbs, vertigo, dimness of sight, profound sadness, and sometimes epileptiform attacks occur. As winter again approaches, the patient again gets better, but his improvement is not so marked as before; the following spring his disease again becomes aggravated, and so every succeeding summer he becomes worse, until at last he sinks with symptoms of low fever. Those who long withstand the disease become demented. The duration of the disease varies from two or three, to ten or twelve years.

Diagnosis.—The limited habitat of this disease, the class of persons to which it is restricted, the time of year at which it is most developed, and the conjunction of the eruption with the general symptoms of this disease greatly facilitate its diagnosis.

Prognosis.—In its earlier stage a favourable issue may be hoped for under the employment of suitable means, but when it has once become far advanced the disease is generally fatal.

Treatment.—This is limited to the treatment of the various symptoms as they arise, on general principles, and to the adoption of hygienic measures.

Warm-baths, a nutritious diet, the avoidance of exposure to great heat, the transportation of the patient to some part where the disease is not endemic. In short—the removal of the causes which we have enumerated above as predisposing to the disease.

MALUM ALEPPORUM.

Synonyms.—Aleppo ulcer; Bouton d'Clep; It. mal d'alepo; Haleb Choban; Habtil Senne; Aleppo evil.

Definition.—Aleppo evil is a chronic disease peculiar to Aleppo and its neighbourhood, occurring only once during life, characterised by the appearance of one or more flat tubercles which ulcerate and leave a cicatrix.

Causes.—The causes of the disease are obscure; it is not inoculable, neither is it contagious. It is endemic not only at Aleppo, but also at Bagdad, and at various cities on the borders of the Tigris and Euphrates.

Description.—The disease consists in the eruption of one or several tubercles. They may appear at any part of the body, but are generally confined to the face and extremities. The face is their favourite situation.

The number of the tubercles varies; there may be only one, or there may be as many as a hundred; generally there are but two or three. When there is but one it is called the "male tubercle." When one or more large tubercles are surrounded by a number of little ones the disease is called by the Aleppians the "female tubercle."

The tubercle at first is very indolent, and is attended neither with pain, heat, nor itching. After spreading for some months without a proportionate increase in height, it at length becomes extremely painful. Suppuration takes place, and an ulcer is found which discharges a thick offensive purulent matter which dries into a scab. The ulcer itself is superficial, uneven and florid, and varies in diameter from half an inch to three or even four inches.

The scab at first is moist and is repeatedly cast off and renewed; but after a few months it becomes dry and adherent, and cicatrization

ace at the end of a year. Sometimes the face is greatly
by the scar.

disease attacks all ages and both sexes. But it appears generally
age of one or two years. It is said that no inhabitant of Aleppo
reached his tenth year without being affected with this "evil,"
marked in the face. It attacks strangers who dwell in Aleppo as
as natives, but then less frequently fixes on the face. Instances
related of Europeans who had resided for a time at Aleppo being
ed many years after they had left the place. The dogs at
are subject to this disease.

nosis.—The disease may be expected to last in all about a
without interfering in any way with the general health, but
ays leaves behind it a scar which is often a very disfiguring

atment.—This is limited to emollient applications, the observ-
of cleanliness, and the occasional sparing use of caustics.

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CORRIGENDA.

Page 35, line 13 from bottom, *for* "all" *read* "although."
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